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PRINCIPLES OF PLANT INFECTION

PFLANZLICHE INFEKTIONSLEHRE
LEHRBUCH DER ALLGEMEINEN PFLANZENPATHOLOGIE
FÜR BIOLOGEN, LANDWIRTE, FÖRSTER
UND PFLANZENZÜCHTER

VON

ERNST GÄUMANN

O. PROFESSOR FÜR SPEZIELLE BOTANIK
AN DER EIDG. TECHN. HOCHSCHULE
IN ZÜRICH

Mit 311 Abbildungen und 90 Tabellen im Text

Eadem mutata resurgo
Jakob Bernoulli, † 1705.



VERLAG BIRKHÄUSER
BASEL

PRINCIPLES OF PLANT INFECTION

A TEXT-BOOK OF GENERAL PLANT PATHOLOGY
FOR BIOLOGISTS, AGRICULTURISTS, FORESTERS
AND PLANT BREEDERS

ERNST GÄUMANN

PROFESSOR OF SPECIAL BOTANY IN THE
FEDERAL INSTITUTE OF TECHNOLOGY
ZÜRICH

Authorized English Edition by

WILLIAM B. BRIERLEY

Professor of Agricultural Botany in the University of Reading



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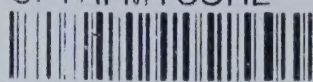
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AUTHOR'S PREFACE

THIS text-book is the outcome of the author's twenty years' teaching in the Swiss Federal Institute of Technology, Zürich. Its aim is to provide an introduction to the principles of plant infection, i.e. to the biological problems which underlie the diseased condition; it does not deal with specialized plant pathology or provide information on individual diseases as such. It is thus a contribution to the theory of plant pathology.

It seeks to describe the general parasitological and epidemiological aspects of plant disease by means of selected examples and to define technical terms, some of which are already in common use. Many of these derive from human medicine but have undergone some change of meaning in their transference to botany; hence, when needful, the differences between phytopathological and medical conceptions are briefly explained.

Naturally, personal judgement has a wide field of choice in the delimitation of subject-matter and the selection of examples; the author is therefore sincerely grateful for the suggestions and advice he has received.

The book was written during the Second World War when Switzerland was an island in the midst of endless suffering, cut off from the mental life and literature of the rest of the world; this is why reference could not be made to some of the newer work.

The English translation has been edited by Professor Brierley with admirable care and I wish to offer him my cordial thanks.

ZÜRICH 6
UNIVERSITÄTSSTRASSE 2
November 1948

PROF. DR. E. GÄUMANN
*Head of the Department of Special Botany
Swiss Federal Institute of Technology*

PREFATORY NOTE TO THE ENGLISH EDITION

PROFESSOR ERNST GÄUMANN'S *Pflanzliche Infektionslehre* was published in the spring of 1946 and later in the year it was my privilege to write a Notice of the book for the *Annals of Applied Biology*. As I have no better words in which to describe its value I may be permitted to quote some of them here.¹

Those who treasure memories of plant hunting in "the joyous Alps" find it easy to understand why Switzerland, a country only twice the size of Wales, with half the population of London, should have produced so many great botanists: "Great things are done when men and mountains meet." Eminent among Swiss botanists is our Honorary Member,² Professor Ernst Gäumann of Zürich. For a quarter of a century notable contributions to many aspects of plant science have streamed from his laboratory and he is recognised the world over as *Magister* in the fields of plant pathology and mycology. One need only recall his *Monographie der Gattung Peronospora* of 1923; his *Vergleichende Morphologie der Pilze* of 1926 which, in C. W. Dodge's excellent translation, has become the mycological Bible of all English speaking students of the fungi; and the *Biologie der pflanzenbewohnenden parasitischen Pilze* written in collaboration with Professor Eduard Fischer of Bern in 1929, an admirable volume which unfortunately has not received translation. And now there has appeared his *Pflanzliche Infektionslehre*, an illustrious work in which twenty years of thought, research and teaching have come to splendid fruition.

It is fortunate that from time to time in the march of science a scholar of deep insight and widely ranging mind brings together in one volume the whole scope of a particular field of enquiry and synthesizes it in such a way that divergent viewpoints and masses of data fall into place to create a unified and harmonious picture. Such a volume materializes the intellectual climate of the time and crystallizes the knowledge into sharp-edged form; it acts as a potent catalyst to workers in its own and adjoining fields and it serves as a master plan for the new era of scientific exploration to which it gives birth. These volumes are rare and still more rarely is it given in the lifetime of one man to produce more than one such book, but Professor Gäumann has achieved this distinction. His new work shows a far-reaching comprehension, a masterly handling of data and an elegance of treatment which derive only from a scholarly mind rich in the wisdom of experience and, running through its pages like golden threads, are a philosophic integration and a processional ordering that endow it with epic quality.

Further, the volume is a notable landmark in the development of the subject. Up to the present the study of plant disease has remained merely an increasing aggregate of data, techniques and expertize relating to its circumscribed area of natural processes and phenomena. It has not attained the status of a science,

¹ From the *Annals of Applied Biology*, 33, pp. 336-7, 1946; by permission of the Hon. Editor.

² Honorary Member of the Association of Applied Biologists.

a "universe of discourse", for such an aggregate only becomes a science in so far as it develops a consistent body of theory which correlates the facts into a logical and explanatory system. The importance of Professor Gäumann's *Pflanzliche Infektionslehre* resides largely in the fact that it is the first serious attempt to develop the necessary theory. It is essentially a treatise on the theoretical foundations of plant pathology and it marks a long and decisive step towards the establishment of plant pathology as a science.

The volume is complete in itself but, at the same time, it overlaps and complements the earlier treatise on the *Biologie der pflanzenbewohnenden parasitischen Pilze*. This dealt with the growth and life-relations of parasitic fungi living in a particular habitat, the living plant. The fungi *qua* fungi occupy the centre of attention and the host plants and other environmental determinants have value only in so far as they provide the conditions necessary for the life of the parasite. In his new book the author makes a critical survey of the entire field of plant invasion by parasitic fungi, bacteria and viruses in relation to the conditions and mutual interactions of the host plants, parasite and environment. It is the relation itself that occupies the focus of attention rather than any one of the three elements involved in the relation, i.e. the state of disease is visualized as an integration at a higher level which subsumes its components. Moreover, infection is considered not as a problem of the special pathology of individual plant diseases but as a fundamental biological problem of the threefold interaction of the constitution of host plants, the invasive potentiality of parasites, and the conditioning factors of the environment. The structures and relations present in individual diseases have value in the author's consideration only as illustrations of the fundamental biological principles. Thus, in his trilogy, Professor Gäumann has laid a splendid foundation for the understanding of the structure and reproduction of fungi, the life of parasitic fungi in their host plants, and the fundamental biological problems created by the host-parasite-environment relationship.

In his *Pflanzliche Infektionslehre* the author's treatment of the subject divides naturally into a sequence of six major topics, each occupying a chapter of the book. Chapter one deals with the manner of entry of a parasite into a host plant and its establishment in the tissues. Chapter two discusses the way in which parasites are brought into contact with their hosts, the influence of the environmental conditions, and the epidemiology of infection. Chapter three considers the virulence and other parasitic qualities of invasive organisms in relation to the genetic constitution and development of the parasites and the conditioning influence of external factors. Chapter four, the longest in the book, discusses the receptivity and defence reactions of the host plant in relation to its genetic constitution and development and to environmental conditions. Chapter five deals with the processes and conditions underlying the morphological and physiological manifestations of the diseased state. Chapter six, a few pages only, indicates the prophylactic and therapeutic principles on which the control of disease is based. The last topic might, perhaps, be considered to lie outside the scope of the immediate problem but, although it receives brief attention, it is treated so suggestively that one is grateful for its inclusion. The first five topics are discussed in great detail and with such a wealth of cogent illustration that more than the bare outline of the author's treatment given above is impracticable in a short Notice. I can only say that I do not remember any other book in the literature of plant pathology that has left on me so strong an

impression of the integrative quality of the author's mind, of his wide-ranging scholarship, or of his powers of luminous exposition.

'It will be little short of a tragedy if this volume does not find rapid translation into English so that it may reach the widest possible circle of readers, for its influence on the development of plant pathology in our time should be incomparable.'

Mr. John W. Wilson, Chairman of the London publishing house of Crosby Lockwood & Son Ltd., saw this Notice in the *Annals of Applied Biology* and decided that an English edition of the book must be produced. He made the necessary arrangements and, as the matter was considered urgent, enlisted the help of Miss Kathleen Secker of the Commonwealth Mycological Institute, Kew; Mrs. F. L. Balfour-Browne, M.Sc., of the Botanical Department, British Museum (Natural History), London; Dr. Joan Moore, then of Rothamsted Experimental Station, later of the Pathological Laboratory of the Ministry of Agriculture and Fisheries, Harpenden; and Dr. W. W. Schwabe of the Research Institute of Plant Physiology, Imperial College of Science and Technology, London.

The work was carried out in the following way. Each chapter of the book was divided into four parts, Miss Secker translating the first quarter, Mrs. Balfour-Browne the second, Dr. Joan Moore the third, and Dr. Schwabe the fourth; as each chapter was completed the scripts were sent to me and the translators moved on to the next chapter. Since the part translations were made simultaneously and independently, the scripts naturally varied greatly in style and treatment, but they were valuable in the quick provision of a working draft of the early chapters whilst the later ones were still under translation. My wife (Dr. Marjorie F. Brierley) and I, working together, then made our own complete translation of the volume using the translators' scripts as a basis but re-writing freely to produce a homogeneous rendering of the original. The final production has, therefore, something of the character of a palimpsest. Corrected proofs were read by Professor Gäumann and certain slight emendations suggested by him in his original text were incorporated. It had been hoped to check all the bibliographic references and to transcribe the abbreviated journal titles in accordance with the *World List of Scientific Periodicals*; part of this task was, indeed, completed by Miss Secker, but it was not found practicable to carry out the original intention and the Bibliography appears, therefore, as in the Swiss edition. The Index to the English edition has been amplified.

In the English text I have retained the scientific names given by Professor Gäumann, which in most cases are the names accepted by the authors whose work he is describing, but I have deleted from the text the appended authors of the names since these appear in the Index. Many of the scientific names used by Professor Gäumann may not be familiar to some readers and I have, therefore, added an Appendix to the book. This includes only those names which are not in common use in the United Kingdom or the United States, names which have recently been changed, or names

concerning which some difficulty may arise in the mind of a general reader; I have added the more familiar synonyms or recent name-changes, the common names of the organisms or diseases, and occasional notes. Although this Appendix is almost entirely nomenclatural it does not pretend in any way to be a critical systematic commentary; it refers only to such points as may help other readers who, like myself, are not systematists, to a fuller understanding of the work. I am grateful to Dr. S. P. Wiltshire and his colleagues of the Commonwealth Mycological Institute for suggesting emendations and improvements in a draft of this Appendix which I submitted to them.

In his book Professor Gäumann introduces many new and useful terms; in translating the book I have needed to coin certain others since, in many cases, the English literal equivalent of the German word was either not meaningful or was already pre-empted. Some of this coinage might be improved, but at the time it was the best I could mint. In order to facilitate cross-reference of the English with the Swiss text I have retained the author's paragraphs although, on occasion, these are not best suited to the English narrative, and I have retained the author's numerical sequences for Tables and Text-figures—even to the omission of Table V, a number inadvertently missed in the original work.

A cynical French writer has likened a translation to his mistress—'belle, elle ne peut être fidèle; fidèle, elle ne peut être belle': but apart from this normal difficulty the translation of a book devoted to the theory and principles of an inexact field of scientific investigation presents innumerable problems and difficulties. Unlike the author's *Vergleichende Morphologie der Pilze* this volume does not consist of factual description; rather is it a fabric of interpretation and closely woven logical argument in which factual description is introduced merely to provide a framework for the author's interpretations and to sustain his arguments and conclusions. Accordingly, Professor Gäumann's personal scientific philosophy, his views and perspectives, the very shape and colour of his words and constructions and every slightest nuance of his meaning, assume an almost exaggerated importance in the translator's task; the translator must, as it were, try to enter the author's mind and view his material through that mind's eye. But the author's selection of data, his balance or emphasis of scientific values in the weighing of evidence, his methods of arriving at conclusions or the conclusions reached together with their implications, may differ very greatly from those of the translator. I doubt whether any botanist or plant pathologist reading Professor Gäumann's volume could agree with everything it contains, and it is a measure of the originality and independence of the author's mind that every reader of his book will wish to argue one or another point with him, will question his judgement in selecting this datum, emphasizing that aspect, drawing the other conclusion, or suggesting a particular implication. A translator is certainly no more exempt from such feelings than any other reader, indeed; rather the contrary since he must ponder each word and thought, and so on almost every page I found myself

wishing to alter or amend the text, to submit further or conflicting evidence, to suggest the possibility of alternative interpretations, or to point to different conclusions and implications. Complete restraint is impossible since, as in the case of the author, a translator's very choice of words is unconsciously determined by his own philosophy, ideas, and viewpoints, and his every turn of phrase shaped and coloured by his personal experiences and scientific perspectives. Hence, in spite of my best conscious endeavours to render the pure Gäumann, I cannot hope to have avoided some glossing of the text.

A further difficulty in the translation arose from the fact that Professor Gäumann possesses a richly imaginative and pictorial mind, and often clothes his ideas and facts in language that is at once more romantic and more teleological than is customary in English botanical writings; he informs me, however, that his words are not to be interpreted teleologically. I have, therefore, either put a transliteration of certain of his words in inverted commas (often followed by the German word italicized in parenthesis) or have used some other word of like connotation but without teleological significance.

Usually, it has not been possible to maintain the author's original constructions, although I have done so whenever feasible, and I have had little hesitation in re-writing freely when this seemed the best way of conveying the meaning of the text to English readers. I have paid attention to the letter but my aim has rather been to preserve the style and spirit of Professor Gäumann's work, to translate as accurately as possible his ideas and thoughts into as simple and readable English as the subject permits whilst, at the same time, trying to re-create something of the atmosphere, the 'mental climate' of the original work.

For a generation to come botanists and plant pathologists will find this book not only a mine of information but a source of inspiration, a true 'Pierian spring'; but they will also find it a mine of argument and controversy and a source of endless discussion and questioning—which is what a book of this kind should be. I can only hope that in making it available to English readers I have not obscured the working faces or muddled the spring.

I wish to accord my grateful thanks to Miss Kathleen Secker, Mrs. Balfour-Browne, Dr. Joan Moore, and Dr. W. W. Schwabe for the knowledge and labour they embodied in the first draft translation; they may not recognize or approve much of the text as it now appears, and for such errors and ambiguities as it contains I must bear the responsibility.

My indebtedness to my wife is not easy to express in words. Without her constant aid in the dire task of translating the entire book and in repeatedly checking scripts and proofs against the original text, this volume would never have come to birth. For eighteen months she surrendered holidays, week-ends, and long evenings to help me in this work and, by rights, her name should accompany mine on the title-page.

My sincere thanks are due to Mr. John W. Wilson of Crosby Lockwood & Son Ltd., whose imaginative courage and generosity made possible the

production of this English edition; his helpful kindness and thoughtful consideration at all times smoothened my way and were greatly appreciated.

Finally, for my wife and myself our task has been a 'labour of love' which we have gladly performed, partly as a token of our friendship for Professor Gäumann and our admiration for his work, and, partly as a tribute to Switzerland, a beloved country which has ever given to us mental and physical refreshment and treasured memories.

WILLIAM B. BRIERLEY

READING

November 1948

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INTRODUCTION

THE condition known in human medicine and in plant pathology as 'disease' cannot be described precisely and exhaustively but it is clear that an essential feature of this condition is the presence of a well-marked and obvious reaction in the affected organism. Where this is absent, except in the case of symptomless diseases, the term 'injury' is preferable. For example, when phanerogamic parasites, such as toothwort (*Lathraea Squamaria*) or semi-parasites, such as eyebright (*Euphrasia*) or cow-wheat (*Melampyrum*), withdraw materials from their hosts without visible reaction, or at most a certain retardation of growth on the part of the individuals attacked, this is not disease but rather weakening or injury.

The manifest reactions of the host are called pathological symptoms and the study of symptoms is known as symptomatology. The nature of a disease is diagnosed from its symptoms and the method of determination is one of differential diagnosis. Etiology is concerned with the causes of disease, therapy with its cure, and hygiene and prophylaxis with its prevention. In medicine the previous history of a case is termed its anamnesis and its course of development its pathogenesis.

Plant diseases are classified as physiological or infectious according to the nature of the agencies which evoke the condition.

Physiological diseases are often negatively termed 'non-parasitic' diseases. They are referable, for example, to internal disorders (such as disturbances of metabolism, formation of growth substances or of organogenic hormones, &c.), to the influence of severe external conditions (e.g. heat, cold, factory fumes, &c.), or to partial malnutrition (deficiency diseases, e.g. phosphorus, nitrogen, potassium, calcium, or trace element deficiency; for the latter see Scharrer, 1941).

The infectious diseases are brought about by contagion and are themselves usually contagious or infectious. The following agents must be considered:

(a) Parasitic fungi (infectious fungal diseases, mycoses). Unlike the infectious diseases of man and other warm-blooded animals which, for the most part, are evoked by pathogenic bacteria and only in minor degree by pathogenic fungi, the latter, for reasons to be discussed later, are the main agents of the infectious diseases of plants. Thus, of 162 economically important diseases of cultivated agricultural plants of central Europe described by Braun and Riehm (1940), 136 (84%) are caused by fungi and only 9 (6%) by bacteria; of some 460 diseases of central European forest trees (Neger, 1924), about 440 (96%) are mycoses and only 20 (4%) are bacterioses. (For a comprehensive account of the fungi see Gäumann, 1926 and 1940, and Fischer and Gäumann, 1929.)

(b) Parasitic bacteria (infectious bacterial diseases, bacterioses). None

of these are spore-formers and no plant pathogenic representatives of the cocci and spore-formers are known with certainty.¹ (An account of the phytopathogenic bacteria is given by Stapp, 1928, and by Elliott 1930.)

(c) Viruses (singular, virus; literally 'poison'; plural, viruses; virus diseases = viroses). Of the 162 infectious diseases of agricultural plants mentioned above, only 17 (10%) are certainly viroses, but their importance for example in potato cultivation, far exceeds this numerical proportion. (An account of the viroses is given by K. M. Smith, 1937; Bawden, 1939; and Kausche, 1942.)

(d) Parasitic phanerogams; e.g. Convolvulaceae (dodder, *Cuscuta*), Orobanchaceae (broomrape, *Orobanche*), and Loranthaceae (mistletoe, *Viscum album*). All these are on the border-line between pathogens and mere weeds.

(e) Parasitic animals (nematodes, gall-forming insects, &c.). In contradistinction to English and Dutch usage (diseases and pests, *ziekten en plagen*), the injuries and occasional diseases produced by animals (entomology) are not always differentiated in German usage from the true infectious and physiological plant diseases (plant pathology); thus, Sorauer's *Handbuch der Pflanzenkrankheiten* (1934-41) covers both fields. The present work is confined to the true infectious diseases: mycoses, bacterioses, and viroses.

The study of plant infection developed during the first half of the nineteenth century out of the conceptual sphere of human medicine (Wehnelt, 1943); thus, Unger, Ehrenberg, Link, Martius, Meyer, Nees von Esenbeck, Persoon, Rudolphi, Willdenow, and De Bary, to mention only the most outstanding, began as doctors. Yet plant pathology has not made scientific advances comparable to those achieved in human medicine.

Primarily this is because of the wide range of its field of study and the consequent temptation to diffusion of work. Human medicine is centred in a single biological species, man, whereas the plant pathologist must concern himself with dozens of economically important cultivated plants and thousands of wild species. Almost every one of these species suffers from dozens, and in some cases from hundreds, of particular diseases. We cannot, therefore, speak of a reaction of plants in general and compare it with a human reaction, since nearly every example in plants can be countered by another reaction in a different kind of plant.

On the other hand, this vast wealth of material increasingly forces the plant pathologist to specialize, but not, like the doctor, in certain organs or organ-groups, e.g. eyes, heart, digestion, or in particular methods, such as surgery, but in distinct species or species-groups, e.g. cereals, potatoes, vines, and so forth, or in definite groups of pathogens, e.g. mycoses, viroses. Moreover, even in the case of an apparently simple object like the

¹ [See Volcani, Z., and Dowson, W. J., 'A plant disease caused by a spore-forming bacterium under natural conditions', *Nature, Lond.*, **161**, p. 980. 1948.]

potato, the known facts of its diseases, which include over 300 mycoses, bacterioses, and viroses, can scarcely be surveyed by a single person.

Plant pathology differs from human medicine not only in the number of species demanding attention but also in the attitude towards the individual. The practising doctor, as also for the most part the veterinary surgeon, is primarily concerned with the individual (individual medicine); even if he be among those influenced by considerations of group medicine, directed towards the welfare of large communities, as in the case of protection from smallpox by vaccination or of mass radiography in the anti-tuberculosis campaign, his methods are concentrated on the individual. When the physician speaks of constitution, he means the constitution of a given individual; for him, idiosyncrasies of reaction are the individual responses of a given patient. This narrow conception derives from the kaleidoscopic potentiality for reaction of the human body (scarcely two persons react alike, so that the course of the reaction can only be predicted in broad outlines), and, further, by the ethical and usually also the economic value of the single human life.

It is different in plant pathology. Personality value seldom attaches to an individual plant, and its economic value, too, is negligible, about one centime for 1.5 g. of the grain and 3 g. of the straw of a wheat plant and four centimes for 0.5 kg. of potato tubers. The anatomical structure of the plant body, compared with that of the human body, is primitive and its reactions correspondingly uniform. Hence, the plant pathologist as a rule takes little account of slight peculiarities in the behaviour of a given plant; unlike the doctor, he does not differentiate on the basis of individuals, but seeks in the course of the reaction of thousands or tens of thousands of representatives of the same systematic unit (race, variety, or species) what is common to all. He is interested in the individuality of the species, not of the single plant; for him, therefore, 'constitutionally conditioned susceptibility to disease' implies the genetically determined susceptibility of particular species, not of particular plants.

In plant pathology, therefore, the individual of human medicine is replaced by the species or race and the objects of its practical measures are entire crops, fields, and the like. Thus, the premisses of plant pathology correspond to those of group medicine. On the other hand, the low economic value and the slight variability of the single plant enable the plant pathologist to carry out his experiments on a large number of similar units and assess the data on a statistically reliable basis with much less difficulty than is the case in medicine.

Notwithstanding these differences in material and premisses, the physician and the plant pathologist are engaged on analogous biological problems; their common fundamental questions are as follows:

1. How does the pathogen penetrate the host? (Chapter I: 'Infection').
2. How does the pathogen gain access to the host? (Chapter 2: 'Infection Chains').

3. What are the conditions for the successful establishment of a disease
 - (a) on the part of the pathogen? (Chapter 3: 'The Parasitic Adaptation of Pathogens').
 - (b) on the part of the host? (Chapter 4: 'The Disease Proneness of the Host').
4. How is the parasitic relationship manifested? (Chapter 5: 'The Disease').
5. How can infectious diseases be combated by man? (Chapter 6: 'The Control of Infectious Plant Diseases').

CHAPTER 1

INFECTION

ALL infectious diseases show a progression through five phases or stages which, in an ideal case, are as follows:

1. Infection. This extends from the germination of the pathogen to the time of its entry into a stable parasitic relationship with its host.
2. Incubation. This covers the parasitologically decisive period between infection and the appearance of the first pathological symptoms, i.e. the 'outbreak' of the disease.
3. The disease proper with its generalized reactions, organic manifestations, and so forth.
4. Healing. The recovery of the organism; healing or the clearance of necrotic tissues from the affected area.
5. Rehabilitation. The restoration of the organism to good health.

Thus, in an ideal example, the course of the disease presents two aspects; infection and incubation lead as an ascending curve to the climax, the disease proper, which is followed by a descending curve of healing and rehabilitation.

The following section deals with the opening stage of infectious diseases, i.e. with infection. This is defined (in the original sense of the word *inficere*) as the conveyance of a disease germ from outside into a host. It covers the attack of a parasite on a host up to its stabilization, that is to say, until a state of equilibrium or at least a close association has been formed between the two partners; only then does the infection 'take'.

In the plant world this phase normally initiates an infectious disease. In human medicine, on the other hand, the situation is much more complicated since here infection does not by any means always directly inaugurate an infectious disease. For example, the pathogenic agent may long have been present in a symptomless and dormant state and the disease may only be evoked by a disturbance in the pathogen-host equilibrium. For instance, there may be a change in the habitat of the pathogen such as that due to typhoid infection of the intestines by way of the bile ducts; or there may be an alteration in its condition, e.g. when existing symptomless diphtheria bacilli suddenly become aggressive and cause immediate illness; or there may be a modification in the host producing a heightened disease proneness, e.g. a 'disposition' to inflammation of the lungs or tuberculosis.

As a rule infection is the colonization by a rudimentary organism, a micro-organism, of one more highly organized, a macro-organism, in which the former grows and multiplies. More rarely, like colonizes like; thus, flowering plants may grow on flowering plants, e.g. Loranthaceae, mistletoes, on broad-leaved trees and conifers, or fungi on fungi. Parasites on other

parasites, e.g. parasitic Fungi Imperfecti on Erysiphaceae (powdery mildews) and rust fungi, which are themselves parasites, are termed hyper-parasites.

When a host that has recovered from an infectious disease is again attacked later by the same pathogen, the term 're-infection' is used. The term 'super-infection' is applied when there is a new infection of an already diseased host by the same or a related pathogen during the existence of the primary infection. The term 'secondary infection' is used when the host is super-infected at this time by an extraneous or different pathogen. A 'mixed infection' is present when a mixture of several parasites jointly penetrates a host.

Six groups of problems are involved in the process of infection in the plant world:

1. How does the pathogen reach the interior of the host? (§ 1. 'The Mechanism of Infection'; p. 6).
2. Under what external conditions does an infection occur? (§ 2. 'The Environmental Conditions of Infection'; p. 19).
3. How long does the infection require to 'take' or establish itself? (§ 3. 'The Duration of Infection, Incubation, and Reproduction'; p. 35).
4. What number of pathogens is necessary for an infection to 'take'? (§ 4. 'The Numerical Threshold of Infection'; p. 38).
5. By what route does the pathogen reach the interior of the host? (§ 5. 'The Path of Infection'; p. 43).
6. How is the occupation of the host effected by the pathogen? (§ 6. 'The Colonization of the Host'; p. 57).

§ 1. The Mechanism of Infection

The mechanism of infection varies to some extent with the nature of the pathogen; whether fungus, phanerogam, bacterium, or virus.

(a) The Infection Process in Fungal Diseases

We may take as a model the infection of a foliage leaf by *Botrytis cinerea*, an unspecialized parasite which in ill-ventilated greenhouses, for instance, causes a putrid dying-off of foliage leaves and young shoots, a grey mould of stone fruits, and a rot of ripe grapes.

Fig. 1, 5 shows a diagrammatic cross-section through the surface of a foliage leaf: *G* ground tissue, *E* epidermis, *K* cuticle of the upper side and cutinized layer of the lower side of the leaf, respectively; upon it a small drop of rain or dew containing the spore *Sp* of a parasitic fungus. Let us assume that, because of high atmospheric humidity owing to dew formation, fog or rain, the drop of water lies for some hours on the cuticle. During this period it undergoes a chemical change because, since the last rain, a transpiration residue of weakly alkaline reaction (pH 7.2–7.8) has been formed on the cuticle, consisting mainly of the carbonates of potassium, calcium, and other salts which diffuse out of the plant body in the

transpiration stream (Arens, 1934; Engel, 1939). A portion of these salts is gradually dissolved in the drop of water, whilst other materials, e.g. phosphatides and growth substances, diffuse in infinitesimal amounts directly from the leaf surface (Arens, 1929). Therefore, the longer the drop remains lying on the leaf, the more does its salt content increase.

These salts furnish a kind of nutrient medium for the spore *Sp*; they stimulate germination and further growth, and the stronger their concentra-

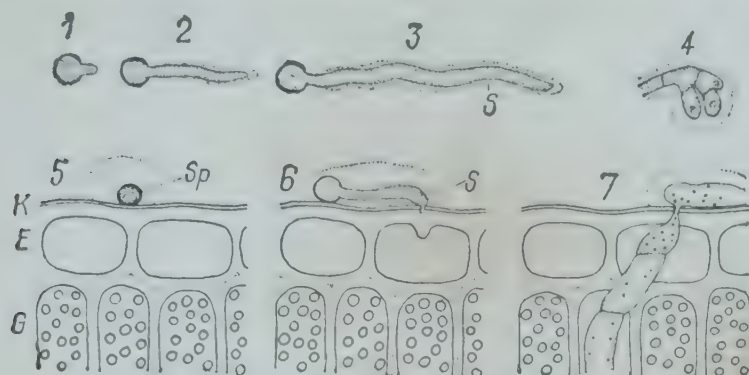


FIG. 1. The infection process in *Botrytis cinerea* on leaves of *Vicia Faba*. Explanation in text. $\times 300$. (After Blackman and Welsford, 1916, and Gäumann, 1933.)

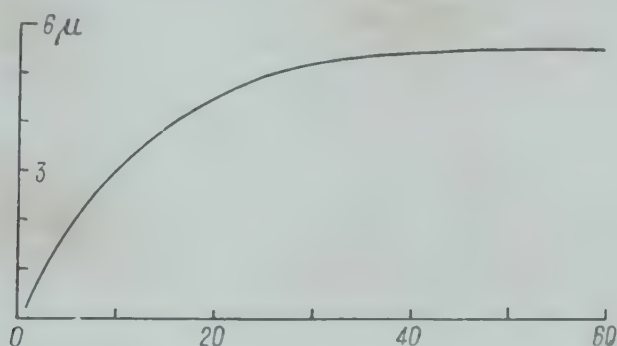


FIG. 2. The stimulation of the parasite (*Botrytis cinerea*) by exosmosed substances of the host (*Cereus 'spectabilis'*, probably *C. speciosus*). Abscissae: electrical conductivity of the water drop in arbitrary units, which, expressed as resistance, are about 10^{-6} Ohm. Ordinates: length in μ attained by the spore germ tubes in 16 hr. (After Brown, 1922.)

tion in the water drop the more the growth-rate of the germ tube is accelerated. In Fig. 2 the electrical conductivity of the water drop served as a gauge for its salt content; in distilled water with a conductivity of 0.8 units the germ tubes grow very slowly; a progressive rise in the conductivity of the drop, i.e. an increase in the materials dissolved in it, is accompanied by a parallel increase in the growth-rate of the parasite up to a certain optimum, at which the germ tubes develop some twenty-five times more rapidly than in pure water.

Thus, the foliage leaf stimulates by its own secretions the germination of a compatible parasitic fungus lying upon its surface which is about to attack it. The same growth-promoting influence on the fungus is exerted, for instance, by volatile or gaseous substances secreted by apples, apple

leaves, &c.; in contrast, the corresponding gaseous secretions of potato tubers, onions, and so forth inhibit its development. Hence, the plant body contributes to its own infection by disease.

An anomalous situation of this kind is alien to human medicine. Thus, sweat, our analogous bodily secretion, with its 1–2‰ of urea and 1–3‰ of sodium chloride, does not stimulate microbial growth; on the contrary, in many cases it inhibits it merely by its acid reaction (acid mantle of the skin).

However, the transpiration residue and the substances which diffuse from it not only incite the growth of the parasitic fungus resting on the cuticle but also attract it by means of a chemotropic physiological stimulus, so that its germ tube now develops towards the plant in the direction of the diffusion gradient of these substances. This, in addition to a negative phototropism, is no doubt the reason why the germ tubes do not wander about haphazard in the water drop but pursue a definite course in the direction of the plant, irrespective of the relative positions of the leaf and the water drop.

Where the hyphal tip meets the cuticle, the tip generally swells and in about 2 to 4 hours forms a disciform organ of attachment, the appressorium. This process is initiated by the mere stimulus of contact, being independent of the nature of the substratum and occurring also, for example, on a glass slide. Lateral branches may be extruded behind the hyphal tip and attach themselves in their turn (Fig. 1, 4), so that the appressorium acquires the form of a hand-shaped tuft. In some other fungi, on the contrary, the appressoria remain small and unbranched.

The wall of the latest formed portions of the hypha swells into a mucilaginous cap (Fig. 1, 5), often glueing the germ tube to the cuticle so firmly that even streaming water, such as a downpour of rain, is powerless to detach it.

With the formation of the appressorium spore germination is concluded and real infection begins, involving the further penetration of the parasite into the host. This, too, is initiated mainly by the stimulus of contact, for the same processes are enacted when *Botrytis* spores germinate on gelatine hardened with formaldehyde or on paraffin wax membranes (Brown and Harvey, 1927).

In the case illustrated in Fig. 1 the germ tube must traverse two layers, the cuticle and the outer wall of the epidermis, in order to reach the interior of the foliage leaf.

The cuticle is mostly about 0.5–1 μ thick and, chemically, consists for the most part of cutin in which plates of wax are embedded. It is practically impervious to fluids, so that the leaf tissue can neither be corroded by enzymes of the germinating fungus which diffuse in advance nor enfeebled by its toxins; the parasite must make its way through the cuticle before it can make any direct contact with the host. Furthermore, the fungi are unable to soften or saturate the cuticle, much less to dissolve it, which means that only very few fungi (and bacteria) are able to decompose substances of the nature of cutin and suberin by means of their enzymes,

i.e. to rot them down (*vermorschen*) like wood. This is very fortunate, for it enables us to leave with safety a cork stopper in our wine-bottles for decades; if beetles do not devour it, fungi can mostly do it little harm even under optimal conditions for their existence.

Thus, the germ tube in Fig. 1, 5, cannot penetrate the cuticle by macerating a small cavity with its enzymes but must rupture it mechanically by force. As we know, it is firmly attached and it now forms a lateral branch, the infection hypha, which perforates the cuticle in the form of an acuminate process with high pressure, up to seven atmospheres. To visualize this achievement we may recall that the average gas pressure in the cylinder of a modern high-powered aeroplane motor engine is roughly seven atmospheres.

On the one hand, the ability of fungi to apply pressures of this order has been experimentally confirmed by direct measurements; Miyoshi (1895), for instance, in a classical experiment, exposed gold leaves, collodion membranes, and the like, to penetration by the germ tubes of *Botrytis cinerea* and demonstrated the pressure applied. On the other hand, the fact of the active perforation of the cuticle emerges from the phenomenon that in certain diseases, as we shall see later, the incidence of infection declines with increasing thickness of the cuticle.

After the infection wedge of the young hypha (in the case shown in Fig. 1) has perforated the cuticle, it meets the second obstacle, the outer wall of the epidermal cell, through which it must also pass in order to reach the interior of the cell.

The cell walls of the plant are about $1\ \mu$ in thickness but as much as $4\ \mu$ on the epidermal surface and in the foliage leaves consist principally of cellulose, a highly polymerized carbohydrate. They undergo further hydration by the secretions of the infection wedge, becoming saturated (Fig. 1, 6) and assuming a lamellate structure. The advancing infection hypha, by means of its enzymes, then dissolves a channel out of the swollen plug, through which it makes its way into the interior of the epidermal cell; beyond the site of perforation it again resumes its full diameter. Using the same mechanism it proceeds from the epidermal cell into the more deeply lying tissues, rupturing the cell walls in turn by the aid of its slender tip, so that in some cases a characteristic constriction develops at the point of entry (Fig. 3).

From the speed with which the hyphae perforate a cellulose wall (about 5 minutes for *Pythium de Baryanum* on potato tubers), it may be inferred that the rupture is not effected purely chemically by the maceration of a pore but that, in addition, the hyphae force a passage mechanically by their own strength (as in the cuticle) through the saturated plug.

In any case, however, the rupture of the cellulosic cell walls of the host

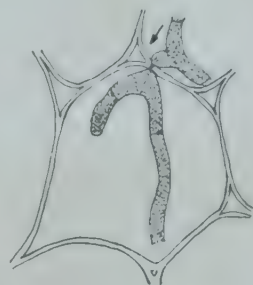


FIG. 3. A hypha of *Gibberella Saubinetii* (seedling disease of wheat and maize) after perforating a slightly swollen cell wall in the mesocotyl. $\times 590$. (After Pearson, 1931.)

implies a new property in the parasite, namely, the secretion of cellulase and of similar cell wall dissolving enzymes. To reach the interior of the cell the fungus must, by means of its enzymes, disintegrate chemically the obstructing cell walls or soften them enough to enable it to push its way through.

The two insulating layers of the foliage leaves, the cuticle and the cell walls, thus embody two distinct selective principles; that of the cuticle is of

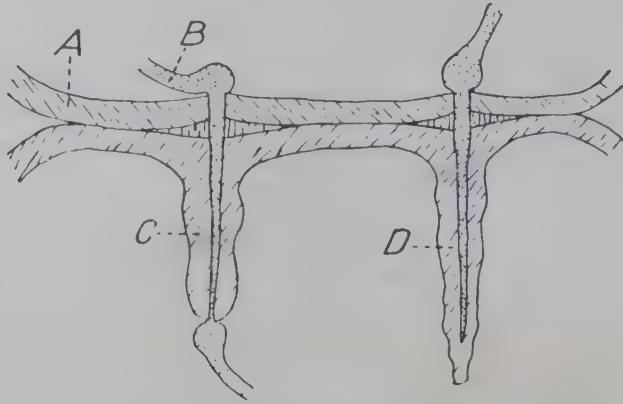


FIG. 4. Lignituber formations in the roots of a wheat seedling after infection by *Ophiobolus graminis* (take-all and whiteheads). *A* cell wall, *B* invading hypha, *C* lignituber perforated by a hypha, *D* lignituber in which a hypha is intercepted. $\times 3,000$. (After Fellows, 1928.)

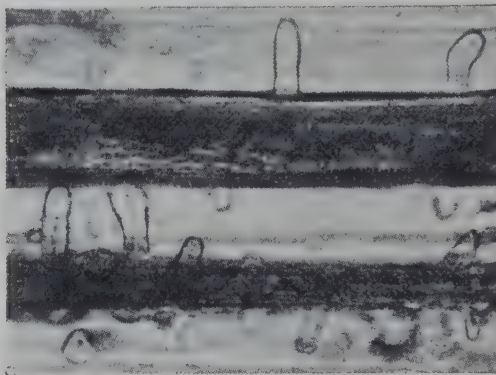


FIG. 5. Lignituber formations in a wheat coleoptile after infection by *Alternaria ribis*. Each lignituber encloses an infection hypha. $\times 360$. (After Young, 1926.)

a mechanical nature requiring a capacity for mechanical perforation, whilst that of the cell walls is chemical as well, requiring a capacity for enzymatic disorganization of cellulose, &c. Only such fungi as germinate in the open on the leaf surface and fulfil both requirements, the mechanical and the chemical, can penetrate to the interior of the leaf by the method illustrated in Fig. 1.

When the infection wedge begins to pierce the epidermal wall the existing dynamic equilibrium of the cell is upset and disturbances ensue within. The pathogen is thus preceded by a sphere of activity, the details of which, however, are little known. Cellular pathology in botany, as in

human medicine, is confronted by the difficulty that the vital processes cannot readily be followed in the cells and, in the little that can be observed on dead, fixed material, cause and effect are often hard to differentiate.

The protoplasm generally undergoes (here we are considering not only *Botrytis* infection) a modification of its structure, which becomes coarsely granular; there is, further, an increase in its power of taking up basic vital colouring matters and a decrease, culminating in reversal, of its plasmolysability. At its plane of contact with the infection hypha it sometimes begins to coagulate or agglomerate, so that the invading parasite is enveloped by a compact sheath or cap which obviously hinders the diffusion of its secretions into the interior of the cell. Through the deposition of hemicellulosic and cellulosic layers these sheaths may be still further

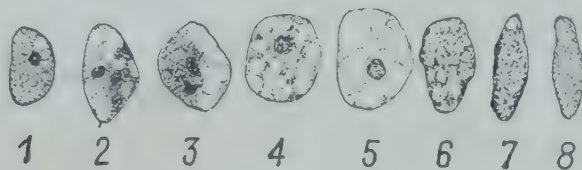


FIG. 6. The degeneration of the host cell nucleus in highly susceptible wheat leaves infected by *Puccinia graminis tritici* (black rust). $\times 750$. (After Allen, 1923.)

strengthened, almost in the nature of wound periderm formation, and converted into real lignitubers (Figs. 4 and 5) which, under certain conditions, are able to seal off the infection hyphae.

The nucleus, also, takes part in the general reaction of the cell. In many cases it migrates towards the infiltrating hypha, being attracted by the wound as the area of maximum physiological activity (traumatotaxis). It applies itself to the infection hypha, becomes applanate, and swells to nearly double its size owing to the enlargement of the nucleolus (Fig. 6, 1). This traumatotactic stimulation can far outdistance the infection hypha; in Fig. 7, for instance, a hypha of *Gibberella Saubinetii* (see p. 9) is penetrating the tissue of a maize seedling from below and has ascended as far as the arrow marked *C*. If the nucleus in the host tissues had migrated to the side of the cell facing the parasite, a dot was made in the first column of Fig. 7; if it lay on the opposite side a corresponding dot was made in the seventh column. The intervening columns represent intermediate cases, so that each dot signifies a host cell. Fig. 7 shows that the nuclei are traumatotactically stimulated, not only in immediate proximity to the hypha but also for a long distance in advance of it. In Fig. 7 its sphere of activity extends over a minimum of twenty cell layers, since a normal distribution of the nuclei only recommences in zone *A*.

Some days later (in Fig. 6) the nucleus of the directly invaded host cell begins to show definite injury and loses its chromatin network (Fig. 6, 5); then it collapses and degenerates on about the fourteenth day into a homogeneous, uniformly staining mass (Fig. 6, 8). A similar fate befalls the plastids, which shrivel and disintegrate.

After overcoming the resistance of the host cells, the fungal hypha forms in their interior specially constructed lateral branches, or haustoria (Fig. 9), shaped like pin's heads, fingers, and so forth, which serve at one and the same time as organs of attack and of absorption (assimilation of nourishment).

Once these have been established, infection may be regarded as assured. A stable relationship now exists between parasite and host; the host's local defences have been surmounted, the process of infection is concluded, and the infection has 'taken'.

The details of the infection process do not, of course, agree exactly in all mycoses with the model example which has been described. They may vary according to the fungus species and the nature and susceptibility of the host. Thus, the infection hypha, instead of penetrating directly into the interior of an epidermal cell, may insinuate itself between the epidermal cells (Fig. 8) into the middle lamellae and only thence extrude its haustoria into the interior of the cell (Fig. 9), i.e. proceed to the actual attack on the host cells.

Moreover, besides direct penetration through the cuticle, there are other channels of infection, such as stomata and so forth, which we shall discuss later (see § 5). In all of them, however, the mechanism of infection is essentially the same: the parasitic fungus actively attacks its host, it develops actively from the infection drop towards the intact surface, and it actively ruptures the cell walls of the tissues. In the mycoses, therefore, i.e. in the most prevalent infectious diseases of plants, the onset of infection has its genesis in a definite assault of the parasite on the host.

Thus, the conditions for infection in the main contagious plant diseases are less favourable than in human medicine. In the science of human infection it is hardly conceivable that the bacteria or spores of tuberculosis, anthrax, or tetanus, conveyed in a drop of water on to our intact skin, should begin to grow and penetrate into our bodies by their independent

activity. The infection process with us, viewed from the angle of the parasite, is normally a passive event and the parasite cannot of its own 'volition' bore a channel from the exterior to the interior of the body. It is drawn into the body passively through apertures or wounds, and only when it has thus passively reached suitable tissue does its development and pathogenic action commence.

To begin with, our bodies owe this initial advantage to the chemical

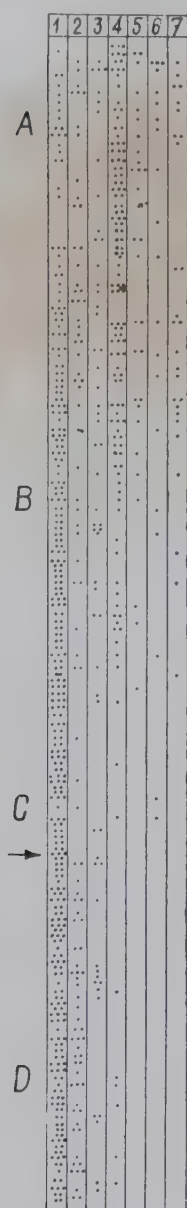


FIG. 7. The traumatic effect of a penetrating infection hypha on cell nuclei. Explanation in text. (After Pearson, 1931.)

protection conferred by the acid mantle, &c., and to the mechanical defence furnished by the horny layer of the skin. It is, however, due in equal measure to the chance that the infectious diseases of the human body, for reasons to be discussed later, are mainly caused by parasitic bacteria rather than by fungi and that the above-mentioned active perforation is normally peculiar to the parasitic fungi.

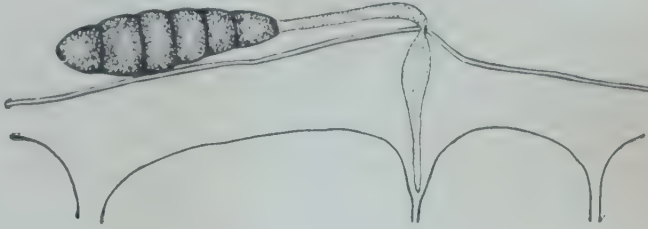


FIG. 8. Germ hypha and infection hypha of *Clasterosporium carpophilum* (shot hole disease) on an almond leaf. $\times 450$. (After Samuel, 1927.)



FIG. 9. Intercellular hyphae of *Cronartium ribicola* (blister rust of Weymouth pine) sending haustoria into parenchymatous cells. $\times 500$. (After Colley, 1918.)

This differential mechanism gives the infection process in plant pathology a different significance from that in medicine. For the practical plant pathologist it is the focus of his studies and precautionary measures since fungi, the habitual agents of plant diseases, can in extreme cases force their way from all sides, e.g. through the foliage, into the plant body. The plant pathologist must, therefore, seek to prevent their germination on the body surface, since, once this has taken place, the infection hypha spreads of its own accord into the interior of the host. Hence the plant pathologist, as a prophylactic measure, covers the entire susceptible body surface, foliage, young shoots, and first buds, e.g. of vines, with a disinfectant such as Bordeaux mixture spray.

For the medical man the situation is different. For him infection is not

crucial to the same degree since his principal pathogens, the bacteria, do not actively penetrate into the organism from the entire, intact body surface. True, the doctor seeks to lessen the risks of infection by hygienic and other measures but the question of covering his patient's whole body with a poisonous substance does not arise; at the most he prescribes a disinfectant gargle, waits until infection has taken hold, and then combats the disease.

It follows from this divergence of viewpoint that the emphasis in the control of infectious diseases (Chapter 6) is differently weighted in the two spheres of activity, the plant pathologist contending primarily with the infection and the physician with the disease.

(b) *The Infection Process in Parasitic Phanerogams*

Infection by parasitic or semi-parasitic phanerogams (Loranthaceae and Tubiflorae) proceeds on similar lines to the attack of parasitic fungi discussed in the preceding section. In many species, such as *Lathraea clandestina*, *Melampyrum pratense*, and *M. sylvaticum*, all three of which are certainly not especially pathogenic, the seeds, like the spores of the parasitic fungi, are incited to germination by the secretions of their appropriate hosts (Heinricher, 1930) and in the really pathogenic species the situation may well be comparable. As in the case of the fungi, parasitic phanerogams show an active mechanism of invasion of the host.

The seeds of mistletoe (*Viscum album*) are carried in the faeces of thrushes, &c., on to the branches of our broad-leaved trees and conifers or are smeared on the bark with the sticky portions of the berry in sharpening the beak. In both cases they are affixed to the branch by the sticky slime (viscin) and germinate, the hypocotyle expanding into an appressorium resembling that of the virginia creeper (*Ampelopsis Veitchii*), which is likewise fastened by the slime. By means of this organ the primary root mechanically perforates the cortex and subsequently the xylem in the form of a haustorium, and successively bursts or cleaves the xylem by its expansion, just as stones are split by roots that have crept into their interstices.

In the following year the primary root forms lateral branches (Fig. 10, 'cortical roots'), which penetrate farther into the cortex sideways and mechanically drive secondary haustoria (Fig. 11) inwards into the xylem as far as the medulla.

Some cells of the haustoria are transformed into vascular bundles (tracheae), and enter into open association with the corresponding organs of the host, from the xylem of which they are supplied with water and salts in solution, whilst the green part outside the host tissues builds up its own carbohydrates and proteins (semi-parasite).

The greater dodder (*Cuscuta europaea*), also, fastens itself to the stems of its host plants in the first instance by an appressorium which, however, is cutinized and hence cannot assimilate its own nutriment. It is perforated by an adventitious root in the form of an haustorium, which penetrates mechanically into the host, crushing the obstructing cells; its apex is

covered by an epithelium the cells of which develop into haustorial cells or haptera. These are long, non-septate cell filaments, which through enzymatic dissolution of the pectins and celluloses advance into the host tissue as

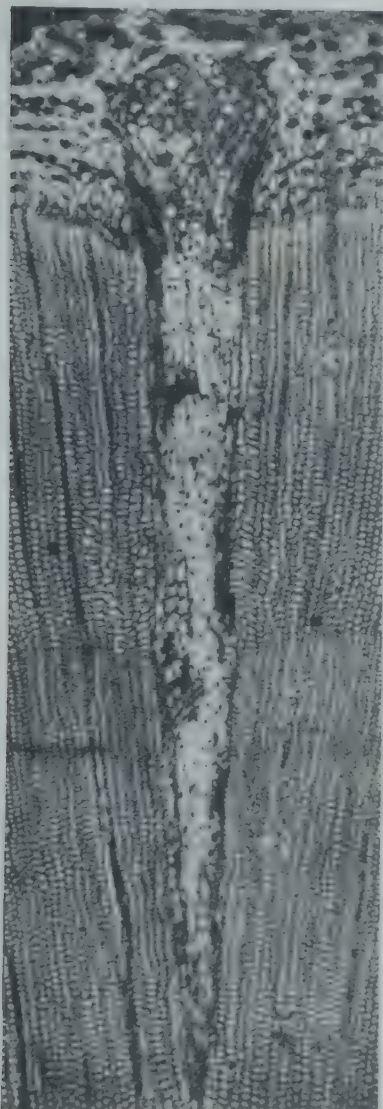


FIG. 11.



FIG. 10.



FIG. 12.

FIG. 10. Diagrammatic representation of the penetration of mistletoe into a host branch. The hypocotyl *h*, transformed into an attachment organ, forms the primary root *1* in the first year, and the lateral cortical roots *r* in the following year, from which the secondary sinkers 2-6 penetrate the main wood *H*. *B* bark, *R* green cortex. (After Neger, 1924.)

FIG. 11. A cortical root of *Viscum album* has penetrated the cortex of a pine shoot (cross-section) and driven a secondary sinker into the wood. Approx. $\times 50$. (After v. Tubeuf, 1923.)

FIG. 12. Haustorial cells of *Cuscuta europaea* in a vascular bundle of *Vicia sepium*. *S* sieve tube, *Z* companion cell, *P* phloem parenchyma. Approx. $\times 150$. (After Zender, 1924.)

far as the vascular bundles, especially the sieve tubes, dissolving and re-absorbing the cell contents, plasma, starch grains, and so on (Fig. 12).

(c) The Infection Process in Bacterial Diseases

In contrast to the foregoing parasitic fungi and flowering plants, the phytopathogenic bacteria are incapable of mechanical penetration of the plant body, cuticle, periderm, &c., since for structural reasons the ability

to form appressoria is lacking. The plant body is, therefore, protected by its cuticle from direct invasion by pathogenic bacteria.

Only two ways of infection are open to these bacteria, namely:

1. An active attack by way of the non-cutinized areas of the plant such as root hairs, stigmas, &c., as well as through injuries, to be discussed later.
2. Circumvention of the cutinized outer layers by passive conveyance of the pathogenic bacteria through apertures from the exterior to the interior of the plant body.

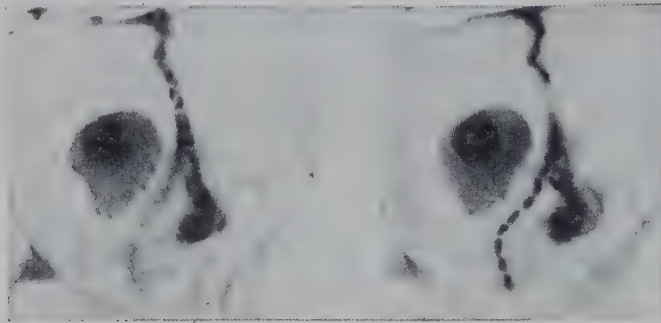


FIG. 13. Chain of bacteria of *Bacterium radiculicola* emerging from an infection thread near a nucleus. Left and right the same structure at different focal depths. Host plant *Neptunia oleracea*, a tropical, aquatic mimosa. $\times 1,350$. (After Schaede, 1940.)

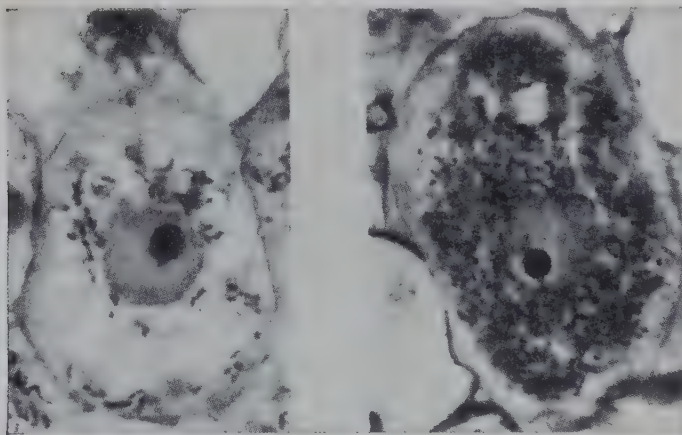


FIG. 14. The multiplication of *Bacterium radiculicola* in the cytoplasm of cells of the root cortex of *Neptunia oleracea*. $\times 900$. (After Schaede, 1940.)

Active invasion by bacteria through non-cutinized surfaces is no doubt feasible only by means of mass infection. *Bacterium radiculicola*, many sub-species and races of which produce root nodules in the Leguminosae (Fig. 178), begins by forming a mucilaginous bacterial colony or zoogloea at the apex of a root hair or, in the lupin, anywhere along the root tip. This dissolves the cell wall and then, in the form of a much curved and branched infection filament or infection 'thread' (Fig. 13), advances into the interior

of the root hair and thence into the cortex of the root almost as far as the endodermis. In most Leguminosae this infection thread is enveloped, like the lignituber (Fig. 4), in a mantle of celluloses and pectins, which is evidently produced by the host cells as a defence reaction and which may lead to complete encapsulation and inactivation of the parasite.

In the fissures of the mantle the bacteria move in chains into the free cell-space where they multiply in the cytoplasm (Fig. 14). The lupin is exceptional in that only the initial stages of infection are carried forward by the infection thread; later, the bacteria occupying the cytoplasm are transmitted from the mother to the daughter cells in the course of mitosis,

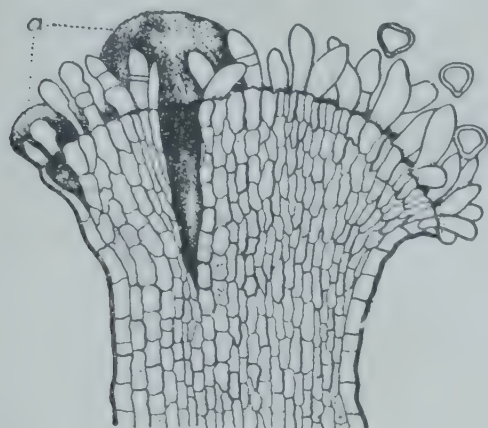


FIG. 15.

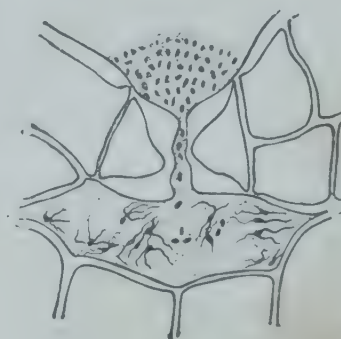


FIG. 16.

FIG. 15. The stigma of a pear blossom with two zoogloea *a* of *Bacillus amylovorus* forcing their way through the style. Diagrammatic. Approx. $\times 180$. (After Hildebrand, 1937, from Leach, 1940.)

FIG. 16. The nectary of a pear blossom infected by *Bacillus amylovorus* through a stoma. Diagrammatic. Approx. $\times 480$. (After Hildebrand, 1937.)

as though they were a part of the normal contents of the cell. Under the stimulus of the bacteria a secondary meristem is formed in the cortical tissue, the division products of which eventually result in the well-known tumours or root nodules (Fig. 178).

In the case of *Bacillus amylovorus*, the cause of the destructive fire blight (*Feuerbrandes*) of fruit trees in North America, the pathogenic bacteria are transmitted by pollinating insects to the stigmas and nectaries of the flowers, where at first they multiply saprophytically. From the stigmatic papillae the zoogloae make their way actively through the inter-cellular spaces of the style (Fig. 15); in the nectaries, which are covered by an epidermis, the bacteria in the nectar drop swarm by their own locomotion through the stomata down into the air chamber (Fig. 16); from both locations in the flower they successively colonize the whole tree.

The passive 'sucking in' of bacteria is effected primarily through the hydathodes and the stomata. Thus *Pseudomonas campestris* (black rot of cabbage) may be conveyed by insects to the leaves where the bacteria enter the guttation drop and, with the return movement of transpiration, are drawn by suction into the cavity *b* of the hydathode (Fig. 17). From

here the bacteria flood into the neighbouring tissue (Fig. 18), where they actively penetrate the vessels and spread through the xylem, and induce chlorosis of the leaves accompanied by a brown or black discoloration of the vein network (Fig. 63).

The pathogenic bacteria are drawn passively through the stomata in dew or raindrops which extend as far as the air chamber in leaves shaken

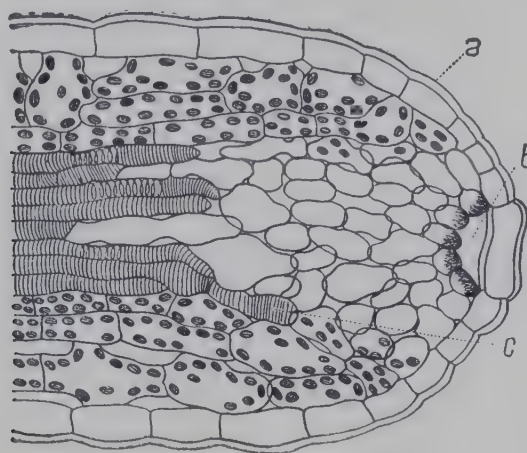


FIG. 17.

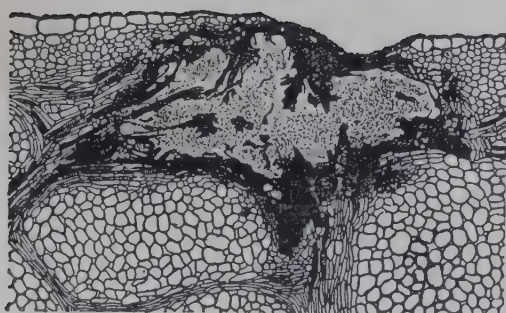


FIG. 18.

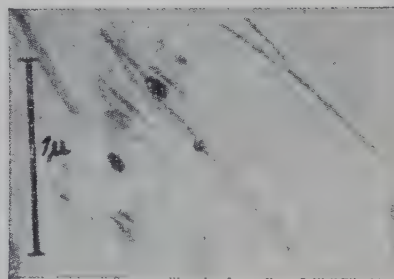


FIG. 19.

FIG. 17. Diagrammatic section through a hydathode. *a* epidermis, *c* termination of the tracheae. Approx. $\times 330$. (After Leach, 1940.)

FIG. 18. Superficial section through the margin of a cabbage leaf invaded by *Pseudomonas campestris* through its hydathodes. Approx. $\times 40$. (After E. F. Smith from Heald, 1937.)

FIG. 19. Electron photo-micrograph of the elongated rods of tobacco mosaic virus. $\times 20,000$. (After Kausche *et al.*, 1939.)

by wind, or with the aid of their flagella they migrate actively down the stoma in the drop and then spread into the intercellular spaces. Thus, the epidemic spread of the wildfire disease of tobacco (*Bacterium tabacum*) requires stormy, wet weather, driving rain, or hail (Böning, 1930). On the other hand, in both wildfire and angular leaf spot of tobacco (*Bacterium angulatum*) the incidence of infection and the area involved on the individual leaf are closely correlated with the width of the stomatal aperture and the water absorption capacity of the leaves (Clayton, 1936, 1937; J. Johnson, 1937; Diachun and Valleau, 1939).

(d) *The Infection Process in Virus Diseases*

Viruses are 'filterable', passing through the pores of the finest inorganic filters, and are sub-microscopically small protein bodies which contain nucleic acid. In the ring spot virus of tobacco (Fig. 223) their shape is spherical, their diameter $19\ \mu\mu$, and their molecular weight approximately 3,400,000; in the tobacco mosaic virus (Fig. 226) they are rod-shaped (Fig. 19), $300\ \mu\mu$ in length, $15\ \mu\mu$ in breadth, and roughly 23,000,000 in molecular weight. They are characteristically able to induce typical diseases in suitable hosts and to provoke the formation of antibodies and, further, they are probably capable of inciting the host cells to form virus protein.

Their inoculation is effected in a purely passive manner, without their own participation, and indeed, as we shall see later, from the mother plant directly into the cell sap of the daughter individuals through injuries or by the agency of sucking insects.

§ 2. The Environmental Conditions of Infection

In human medicine the development of most pathogens begins only after they have reached suitable tissues of the oral cavity and interior of the body, where they settle under optimal, constant conditions of existence, e.g. of temperature and humidity. Hence, the mechanism of colonization of the host by the parasite is seldom materially affected by environmental factors like the weather, the influence of which acts for the most part indirectly on the disposition of the host to disease.

It is otherwise in plant pathology. Here, only the germs transmitted in infection through wounds are immediately enclosed within the attacked plants and so withdrawn from the influence of the external environment. The remaining pathogens begin their development on the surface of the plants they are about to invade, i.e. virtually in the open, since plants have no body temperature of their own. Under these exposed conditions their first step must be to germinate, so that from the outer world they can penetrate into the host. The mere presence of the causal organism, therefore, does not suffice for an outbreak of disease, since certain external requirements must also be fulfilled before the pathogen can begin to develop any activity.

The significance of environmental conditions for infection is, therefore, quite different in human medicine and in plant pathology. In man there must be a disposition, induced by the environment, before the infection can 'take', whereas, in plant diseases, external conditions must be such as to allow of the penetration of the host by the pathogen; in the plant, therefore, disposition has a much slighter role than it has in man.

Accordingly, in most plant diseases, the *conditio sine qua non* for infection is that environmental conditions permit the germination and initial development of the pathogen; a group of environmental factors external to the parasite-host relationship thus determines the occurrence of that relationship.

Environmental factors, of course, modify susceptibility to disease in

plants as they do in man (see 'Disposition'; Chapter 4), but before the question of disposition even arises they allow or forbid the occurrence of infection. Since the most important factors, humidity, temperature, and so forth, are in their turn regulated by the weather, the latter is of direct and critical significance for the incidence of infection in plant diseases. In some of them, e.g. downy mildew of vine, the incidence of infection can sometimes be predicted from the course of the weather.

Conversely, in certain other plant diseases, the environmental factors which are biologically important coincide only partially with the obvious meteorological and edaphic or soil conditions, since the apparent conditions are critically modified in the immediate vicinity of the leaf or root. For instance, the herbaceous canopy over a potato field or a hop garden (Zattler, 1932), forms beneath itself a new and special local or micro-climate (Geiger, 1942), out of 'tune' to some extent with the more general meteorological conditions; similarly, roots surround themselves with an individual rhizosphere. In these cases the macro-climate provides only the general situation within which the micro-climate is further differentiated; within the latter infections will be only indirectly influenced by changes in the weather.

From the complex of factors which together make up the environment we can select and analyse only those which are amenable to physico-chemical definition, e.g. humidity, temperature, availability of light, carbonic acid content of the air, and the acidity of the germination solution.

There are, however, many other factors which, at present, we cannot isolate and which, therefore, we term *imponderables*. Atmospheric pressure may be taken as an example: thus, the zoosporangia of *Phytophthora infestans* (potato blight, Fig. 195) germinate only half as prolifically when the Föhn wind blows as at normal barometric pressure (Fischer and Gäumann, 1929). The development of the saprophytic micro-organism *Azotobacter chroococcum*, and its powers of nitrogen fixation, also appear to depend on atmospheric pressure, being stimulated by an approaching rise and inhibited by an imminent fall (Bortels, 1940).

Conversely, a stimulatory long-distance effect may sometimes emanate from living tissues, notably when the vitality of the pathogen is at a low ebb. Fragments of any kind of plant tissue introduced into Petri dishes containing slides on which brandspores of *Ustilago zaeae* (maize smut, Fig. 144) have been laid to germinate, increase the germination rate from about 40 to 60–80% (Platz *et al.*, 1927). The germination of the basidiospores of Hymenomycetes, which often presents great difficulties, can sometimes be expedited by placing a fragment of the fruit body concerned in contact with the drop of liquid (tissue culture method; Ferguson, 1902).

Washed soil promotes the germination of the spores of *Tilletia tritici* (bunt of wheat), either through an as yet undefined ionic action of the soil solution or through synergism with other micro-organisms (Volkart, 1906). In the assay of plant protectants it is advisable, therefore, to conduct the slide germination tests on a thin layer of washed soil.

It is a task for future research to elucidate the decisive elements in all these *imponderables*.

However, even if we restrict our attention to the definable factors mentioned above, their influence on the establishment of infection becomes ever more complex the deeper the analysis proceeds. In the first place, they determine the antecedent conditions under which the pathogen germinates and penetrates into the host; it is this aspect with which we are concerned here. At the same time, however, they also influence the vitality and the parasitic adaptation of the pathogen and the disease proneness

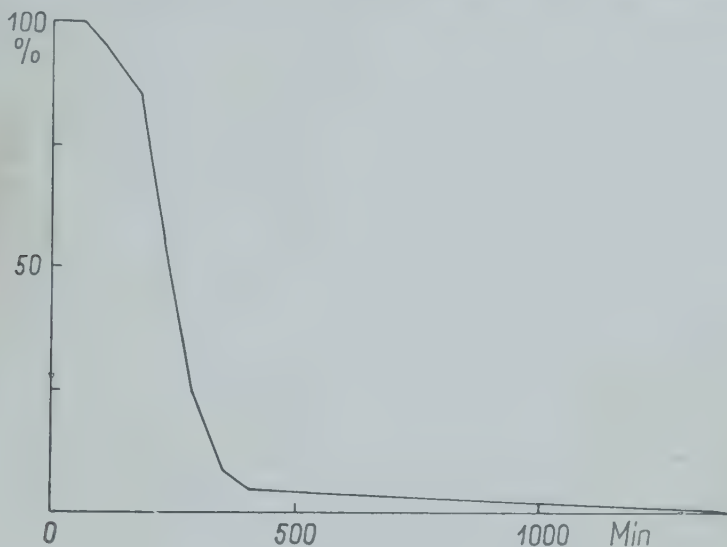


FIG. 20. The influence of moist heat (37° C.) on the germination of conidia of *Botrytis cinerea*. Abscissae: time of exposure in minutes. Ordinates: % of conidia surviving treatment, i.e. still germinable. (After Henderson Smith, 1923.)

of the host (see Chapters 3 and 4). These diverse effects are inextricably interwoven and are sometimes mutually antagonistic.

Moreover, the influence of the environment on the germination and growth of the pathogen is itself complex. In the first place, all spores do not respond with exact uniformity to a given factor, but preserve a certain individuality in relation to it. The first difficulty, therefore, is in the methodological field: what do our statistical data actually express?

As an illustration let us take the influence of rising temperatures on the germinability of the conidia of *Botrytis cinerea*. Let us ask the question: Where does the thermal death-point lie for conidia in a humid atmosphere? Slides covered with nutrient agar are seeded with conidia and, in the case illustrated in Fig. 20, placed in a thermostat at 37° C. After a certain period (abscissae) a number of slides is transferred so that germination is at the optimum temperature of 24–25° C. Although a period of 60 minutes in the thermostat induces no perceptible damage, yet after 23 hours treatment spores can no longer germinate.

When the same test is repeated at higher temperatures, the periods of influence are increasingly curtailed (Fig. 21), a 2-minute exposure to 44.3° C. sufficing to reduce germinability to 96.2%, and one of 40 minutes to reduce

it to 0.1%. After a 2-minute exposure to 50.3° C., only 0.3% of the spores survive, after 5 minutes only 0.05%, whilst a period of 6 minutes is 100% lethal.

What, therefore, is the thermal death-point of the conidia of *Botrytis cinerea* in a humid atmosphere? Obviously three zones must be distinguished, beginning with the favourable vital zone, in which the conidia can remain indefinitely without incurring injury; its upper limit lies around 33° C. This is followed by a zone of thermal instability within which the conidia show reduced viability or injury, although recovery is possible if

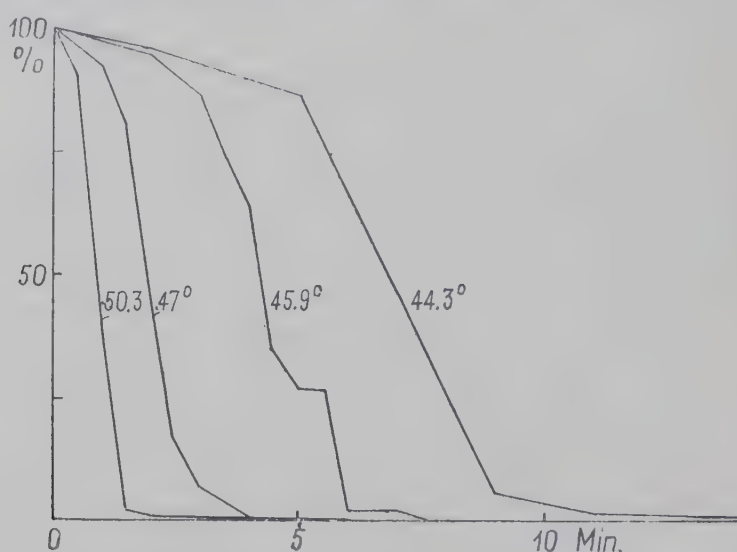


FIG. 21. The influence of moist heat on the germination of conidia of *Botrytis cinerea*. Abscissae: time of exposure in minutes. Ordinates: % of conidia still germinable. (After Henderson Smith, 1923.)

they are returned to favourable temperatures; this zone extends from about 33 to 36° C. Temperatures above this form the lethal zone in which permanent injury becomes evident. The lethal effects show a certain gradation; at 37° C. the injurious factor prevails only with long exposure; with higher temperatures the reaction time becomes shorter and progressively more conidia perish so that the curve, as shown in Fig. 21, becomes steeper. When the absolute lethal temperature limits are reached all the conidia die instantly.

Therefore, in spite of the apparent simplicity of the question, it is not possible to state precisely the lethal temperature limits of *Botrytis cinerea*. It is not a matter of a single degree or point, not 37° C., for example, but of a wide range of temperature extending between 37 and 51° C., within which other factors such as period of exposure, &c., are decisive.

Similar methodological difficulties arise in fixing ecological cardinal points, especially maxima and minima. As a rule, mean values will suffice, but these are mostly inadequate in epidemiology where the highest and lowest values are decisive; for example, the most rapid rate of spore germination or the maximum spore resistance.

Again, the effect of single external factors on the germination and growth of a pathogen is often difficult to assess because the conditions which favour

germination may not be those which immediately initiate the process; that is to say, the internal conditions required to initiate germination may be different from those necessary for its continuance. Thus, the growth substance requirements of a fungus may change during its development; for example, *Rhizopus suinus* requires meso-inositol only during spore germination and the early stages of mycelial development and not subsequently (Schopfer, 1942). It would appear that, in its earliest stages, the fungus either cannot manufacture the growth substance or that it cannot do so in sufficient quantity and that it must, therefore, obtain it from

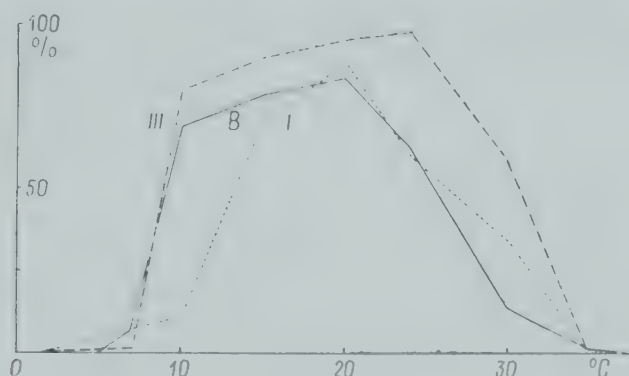


FIG. 22. The influence of air temperature on the germination of the acidiospores (curve I), teleutospores (curve III) and basidiospores (curve B) of *Gymnosporangium globosum* (cedar rust of apple in the eastern United States). (After MacLachlan, 1936.)

the substrate; at a certain stage in its development either it becomes able to synthesize the material for itself or it ceases to require it.

Further, the germination of many resting spores, mostly over-wintering spores such as the teleutospores of rusts or the brandspores of smuts, is stimulated by alternate freezing and thawing, or alternate wetting and drying. But the thermal optimum, following successful stimulation, is about 16° C., i.e. a very different optimum from that required to stimulate germination. Again heat treatment, for example 20 minutes at 52–54° C., can break the resting period of ascospores and stimulate germination (Goddard and Smith, 1938). For optimal infection, therefore, the climatic factors must supply first the conditions necessary to stimulate germination and then the conditions for its progress.

In addition the climatic requirements may vary considerably for different spore forms of the same parasite, e.g. the oospores and conidia of downy mildews or the acidio-, uredo- and teleutospores of the rusts (Fig. 22) and because of this the climatic relationships of the parasite become still more complex.

Again, the external factors which control the percentage and the speed of germination and the rate of vegetative growth of the parasite differ. For example, in *Clasterosporium carpophilum*, which causes a shot hole disease of stone fruits (Fig. 209), there is little correlation between air temperature and the amount of germination; from 90 to 96% germination

will occur within the wide temperature range of 9 to 27° C. (Fig. 23). However, the speed of germination in the same fungus shows a pronounced optimum at 18.4° C. (Fig. 24), and an equally definite optimum for vegetative growth at 21.8° C. (Fig. 25). For optimal infection it is essential that the external temperature during germination and the early stages of growth of the germ tubes should be lower than for the vegetative spread

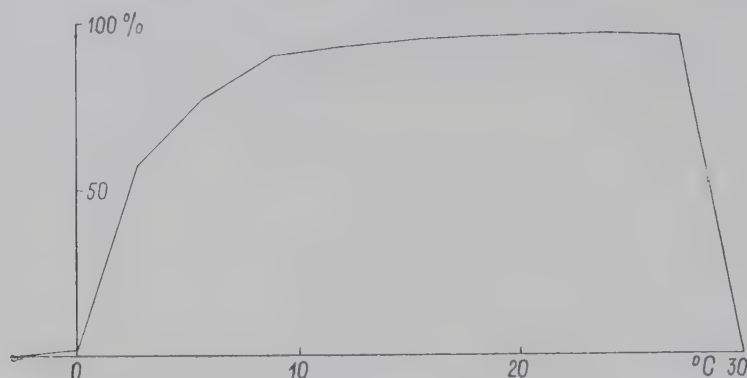


FIG. 23. The influence of air temperature on the germination of conidia of *Clasterosporium carpophilum* (shot hole disease). Abscissae: temperature of germination. Ordinates: % of spores germinating within 10 days. At 30° C. germination still occurs but the germ tubes soon die. Orig. (After St. Roth.)

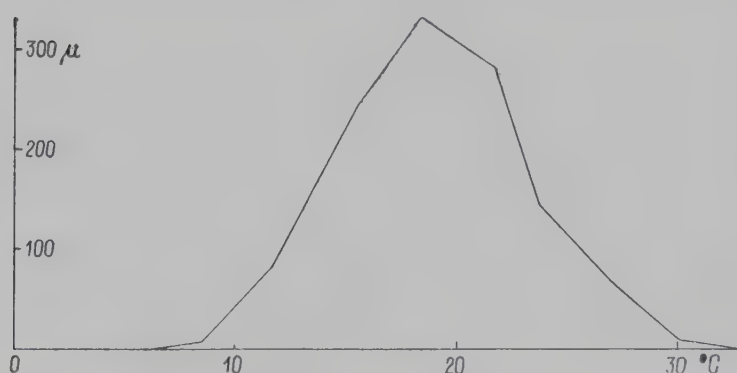


FIG. 24. The influence of air temperature on the speed of germination of conidia of *Clasterosporium carpophilum* (shot hole disease). Abscissae: temperature of germination. Ordinates: length of germ tube, 30 hours after germination has begun. Orig. (After St. Roth.)

within the host, as afforded, for example, by night and day temperatures respectively.

Although a spore may be able to germinate and to put out a germ tube it does not necessarily follow that it is able to cause infection. For instance, a parasite has little vitality under conditions approximating to the extreme limits of its existence, i.e. in the vicinity of the maximum and minimum values, and a spore, therefore, whilst still capable of germinating might not be capable of penetrating its host. The temperature range conditioning infection is narrower (as will be shown more fully in Chapter 3, 'The Parasitic Adaptation of Pathogens') than that determining the general life processes, e.g. growth, metabolism, &c. The limiting values above or below which

infection can no longer take place are known as the critical points; thus, *Bacterium tumefaciens* can exist, though with difficulty, at 37° C., but its critical temperature lies between 28 and 30° C. and, above this, infection can no longer take place.

None of these data has the character of an absolute value, since each holds good only for the particular conditions obtaining at the time of its estimation. The value of a factor will change as a result of its interaction with all the other factors, external factors being, to a certain extent, interchangeable (interference effects, substitution factors). Thus, the temperature curve shown in Fig. 24 holds only for the optimum conditions of germination, namely, free, ionized water having an acid reaction. Should one of these

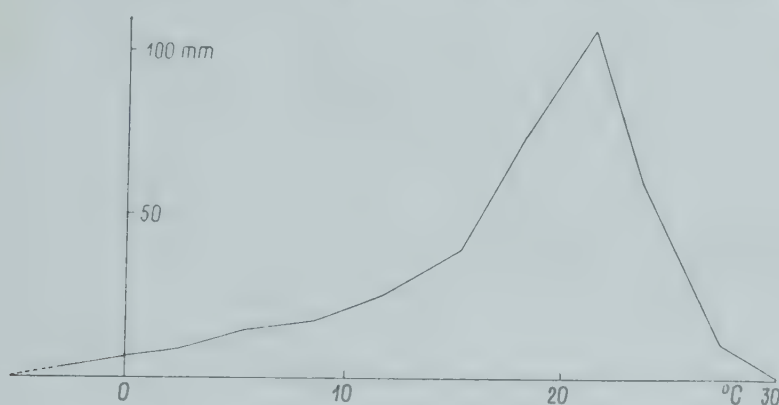


FIG. 25. The influence of air temperature on the vegetative growth of *Clasterosporium carpophilum* (shot hole disease). Abscissae: temperature of growth. Ordinates: diameter of cultures on malt agar after 30 days. Orig. (After St. Roth.)

factors become sub-optimal, as often happens in nature, then the temperature limits will also change. This holds true also for the result of infection since, if moisture conditions are optimal, then the ensuing infection may be severe even at a sub-optimal temperature; on the other hand, a sub-optimal temperature can be counterbalanced by a massive infection, and so on.

Because of such complications and discrepancies results obtained in the laboratory cannot be expected to hold good, without further adjustment, when applied to field conditions. One thing only is certain, that no infection will ensue outside the limits established by laboratory tests. But it is difficult to ascertain exactly how individual factors interact within those limits or what happens at the limits, especially, and it is important to emphasize this, as climatic and edaphic factors influence not only the intensity of the parasite's activity but also the intensity of the host's reaction.

In consequence of these various limitations and difficulties, the actual infection occurring at any time is only a small percentage of what is theoretically possible. This is not only on account of the powers of resistance of the host and the relatively small chance of the spores falling upon receptive organs of susceptible plants, but particularly because of the limitations set by the requirements of infection.

If a pathogen makes only small demands on the environment and can

produce infection within a wide range of ecological conditions it is termed 'euryzoic'. On the other hand, if a pathogen is highly selective and the ecological conditions under which it can bring about infection are very restricted, it is termed 'stenozoic'.

In the following sections the effect of (1) moisture, (2) temperature, (3) light, and (4) hydrogen-ion concentration on the process of infection is discussed. The influence of these external factors on the viability, reproduction, and parasitic adaptation of the organism, on the disease proneness of

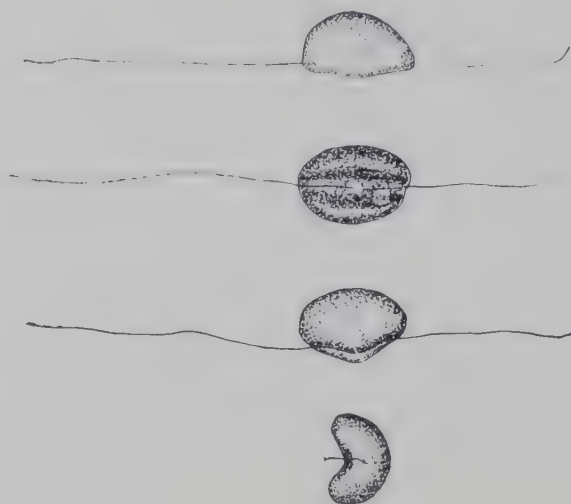


FIG. 26. Zoospores of *Plasmopara viticola* (downy mildew of vine).
Approx. $\times 800$. (After Arens, 1929.)

the host, and on the course of the parasitic relationship will be dealt with subsequently.

1. *The Influence of Moisture on the Process of Infection*

Spore germination and the early stages of the development of a pathogen are, in the majority of parasitic plant diseases, closely bound up with moisture relations: therefore, a sufficiently humid atmosphere in the case of aerogenous or air-borne infections, and sufficiently moist soil in the case of chthonic or soil fungi, is an essential pre-condition for the occurrence of infection.

Infection by air-borne parasites occurs optimally in drops of water, whether rain, mist, or dew (cf. the diagram in Fig. 1). Free water is particularly important for flagellate bacteria, and for the myxamoebae or zoospores of *Synchytrium endobioticum* (wart disease of potato), *Pythium de Baryanum* (damping-off, e.g. of sugar beet seedlings, &c.), *Phytophthora infestans* (potato blight), and *Plasmopara viticola* (downy mildew of vine, Fig. 26), &c. However, under sub-optimal conditions, the conidia of downy mildews can germinate by means of germ tubes, i.e. they can germinate directly instead of by the indirect method of producing zoospores which must first surround themselves with a cell wall before they can put out a germ tube.

Dew, especially, plays a more significant role in epidemics than is generally recognized. Thus, following transpiration the foliage may become under-cooled by some tenths of a degree owing to expenditure of heat in vaporization. When, after sunset, direct heat irradiation ceases and the air temperature falls under a clear sky (night radiation) the saturation point on the leaf surface may be exceeded and, in a still atmosphere, condensation of water vapour will occur. The deposition varies with the angle of inclination of the leaves, with their mutual overlap, &c., but in central Europe, after a night of heavy dew, it may be as much as 0.1 mm. (Jaag, 1945); in the Tropics there will be still more.

In central Europe during the active growing season about one-half of the nights show a heavy fall of dew so that, under the climatic conditions of this region, dew is the most frequent source of moisture for infections. This is recognized by all collectors who find that parasitic fungi are most abundant in the regions of discharge of lateral valleys and watercourses, because it is here that descending currents of cool air lead, almost nightly, to a heavy deposition of dew.

It is true that dewdrops last for only a short time, perhaps 5–8 hours, but since infection can be achieved within this period by means of propagative spores (as will be shown in § 3, 'The Duration of Infection'), the dew lasts long enough for most parasitic plant diseases to develop new centres of infection. In the cases of potato blight and downy mildew of vine, four hours of dew and a temperature above 10 °C. will allow infection to 'take' because it is the most rapidly germinating spores that are decisive.

Not only rain and fog but fine weather also, such as still or warm, brilliant summer nights, may provide ideal conditions for plant infection. Thus, on account of rain in bad weather and of dew in fine weather, our cultivated plants are always liable to infection.

Should it happen that no drops of water are available to the aerogenous pathogens it is still possible for the germination of propagative spores to proceed sub-optimally, i.e. in a moist atmosphere, but the humidities required to correspond to the different germination temperatures for this type of infection are not known with certainty.

It is difficult to estimate the limiting values of humidity in spore germination since they vary with the type of substrate. As is shown in Table I, germination on microscope slides falls abruptly at a relative humidity of 96%; on the other hand (Table II), when foliage leaves are used, germination is still high at a relative humidity of 24–22%. The results, therefore, vary according to the different hygroscopic qualities of the two substrates and also depend on cuticular and, in part, on stomatal, transpiration. These two factors tend to produce a more humid micro-climate in the layer of air in immediate contact with the leaves and, therefore, germination can take place at meteorological humidities lower than that at which it is possible for it to take place on a glass slide.

Again, the determination of the humidity at which infection is still practicable is rendered more difficult by the discrepancy (already mentioned

on p. 24), between possible spore germination and the additional vitality required to allow infection to proceed. When, for other causes, the process of infection meets with difficulties, then it can continue successfully only if the humidity of the atmosphere be optimal. Alternatively, if infection takes place readily, the humidity may deviate from the optimum. Thus, to infect old or mature vine leaves with downy mildew, a relative humidity of 80–100% is necessary, whereas for young leaves 70–80% will suffice.

TABLE I

The influence of atmospheric humidity on the germination of the conidia of Sphaerotheca pannosa (rose mildew), on microscope slides at an air temperature of 25.2° C. (After Longrée, 1938.)

Relative humidity	Germination
%	%
99.0	97.8
98.0	90.4
96.9	98.4
94.9	2.8

TABLE II

The influence of atmospheric humidity on the germination of the conidia of Sphaerotheca pannosa (rose mildew), on detached leaves of Excelsa roses at an air temperature of 21° C. (After Longrée, 1938.)

Relative humidity	Germination
%	%
98–95	99.2
93–91	87.5
89–85	82.0
79–76	68.0
51–48	50.3
30–28	53.5
24–22	37.5

Hence, the lower limits of humidity at which infection is still possible under various different circumstances must be ascertained for each individual disease. Thus, *Cercospora beticola* (leaf spot of sugar beet) needs a relative humidity of at least 98%, approximately saturation point (Schmidt, 1928); germination ceases even at 97%. At a relative humidity of 98% the under-cooling of the leaves effected by transpiration will lead to condensation round the spores and germ tubes, so that, in fact, they develop in actual drops of water; the foliage, therefore, need not be drenched for infection to occur.

On the other hand, *Cladosporium fulvum* (leaf mould of tomato, Fig. 177) already germinates freely at a relative humidity of 90%. Therefore, if the disease in greenhouses is being controlled indirectly by regulating humidity, then the latter must not rise above 80% at a temperature of 18° C.,

while, with a temperature of 22° C., humidity must not exceed 70% (Small, 1930). This is, therefore, an expensive process since it is difficult to maintain a low humidity in a greenhouse without a considerable loss of heat.

For air-borne parasites the optimum humidity coincides with the maximum, namely water in actual drops and, therefore, for these pathogens conditions can never be too humid. It is different for chthonic or soil organisms. Too high a water content of the soil is injurious to pathogenic micro-organisms because it impedes their respiration, favours saprophytic competition, and accelerates the development of the host. On the other hand, a water content that is too low retards or even prevents germination of the spores of the parasite. Thus, bunt of wheat gives an infection incidence of 55.3% when soil is ordinarily moist, with a water content of 40%; of 10.7% in very damp soil, with a water content of 80%; and of 22.3% in very dry soil, with a water content of 20% (Rabien, 1927). In contrast to aerogenous organisms the moisture requirements of the chthonic type distribute themselves about a normal bilateral curve with a minimum, an optimum, and a maximum.

2. *The Influence of External Temperature on the Process of Infection*

The significance of external temperature for successful infection is much less than that of the humidity factor. Whilst moisture is the essential precondition, temperature, on the other hand, can be regarded as having only a differentiating effect, conditioning retardation or acceleration.

In the literature dealing with the cardinal points of temperature, minimum, optimum, and maximum, contradictions are sometimes found owing to the fact that spore material of different ages has been used. Young, highly viable spores have, as a rule, the greatest capacity for germination and are correspondingly little affected by the temperature; hence, curve *I* in Fig. 29 is very shallow. Older spores gradually lose their vitality and powers of resistance and make greater demands on their environment (Fig. 29, curve *II*). Here again, as in the case of the humidity curves, it is a question not of absolute values but of data which hold to a certain degree only, i.e. they are valid only for the conditions prevailing during the tests.

It should be noted that the 'maximum' here refers to the thermal limits of germination and growth, and not to the thermal limit of death; the latter is probably the same for phytopathogenic bacteria as for bacteria which are saprophytic or pathogenic to man. As regards plant pathogenic fungi, this point has been little investigated because killing of spores by heat, as in thermal disinfection or sterilization, scarcely comes into the field of plant pathology; in temperature treatments by warm water or hot air, for example, it is usually only mycelium inside the host which is killed.

In relation to infection the external temperature is important in three ways: firstly, in its effect on the percentage of germination; secondly, in its effect on the rapidity of germination; and, thirdly, in its effect on the growth rate of the germ tube. Its influence on the vitality and aggressiveness of

the pathogen will be dealt with in Chapter 3 ('The Parasitic Adaptation of Pathogens').

The influence of external temperature on the percentage germination of spores is highly variable. The conidia of *Clasterosporium carpophilum* (Fig. 23) are thermally non-responsive and euryzoic, and germination up to 90–96% can occur within a wide range of temperature, e.g. between 9 and 27° C.; only in the vicinity of the minimum and maximum does the percentage germination fall steeply. The germination of the uredospores of cereal rusts is also very little dependent on temperature (Fig. 27), but the

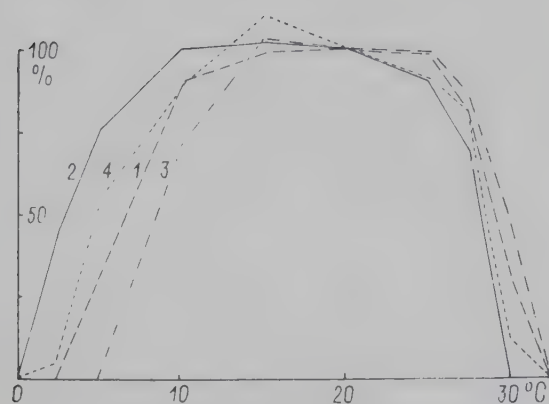


FIG. 27.

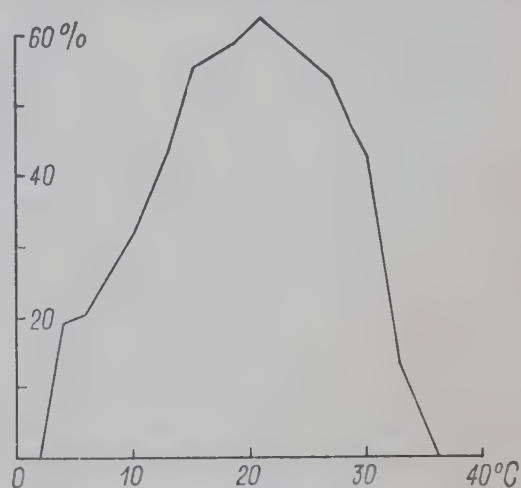


FIG. 28.

FIG. 27. The influence of air temperature on the germination of the uredospores of some cereal rusts. Curve 1: *Puccinia tritici* (brown rust of wheat). Curve 2: *P. dispersa* (brown rust of rye). Curve 3: *P. lolii avenae* (crown rust of oats). Curve 4: *P. graminis tritici* (black rust of wheat). (After Stock, 1931.)

FIG. 28. The influence of air temperature on the germination of the oidia of *Sphaerotheca pannosa* (powdery mildew of rose). Duration of germination experiments 48 or 96 hours. (After Longrée, 1938.)

optimum for crown rust of oats is invariably higher than that, for instance, of brown rust of wheat.

On the other hand, the percentage germination of the oidia of *Sphaerotheca pannosa* (powdery mildew of rose) is closely related to air temperature (Fig. 28), showing a minimum value at 3° C., an optimum between 18 and 24° C., with a distinct peak above 21° C., and a maximum at 36° C.

In contrast to the percentage germination of spores, the rapidity of their germination in all pathogenic fungi is closely associated with external temperatures. The minimum temperature is generally about 1–2° C. and the maximum about 30–36° C. The optimum, on the other hand, often differs characteristically in different species.

Thus, *Pseudoperonospora humuli* (downy mildew of hop) is a relatively low-temperature fungus. Its conidia germinate with optimum speed at 17–18° C. (Fig. 29) but they are still able to germinate at temperatures between 0 and 1.5° C., although they then take approximately 20 hours as against 2–3 hours at the optimum temperature. The zoospores, on the other hand, retain their vitality longer at low than at high temperatures

(Fig. 30). The upper limit for conidial germination and for zoospore swarming, about 30 °C., lies considerably below the air temperature in the

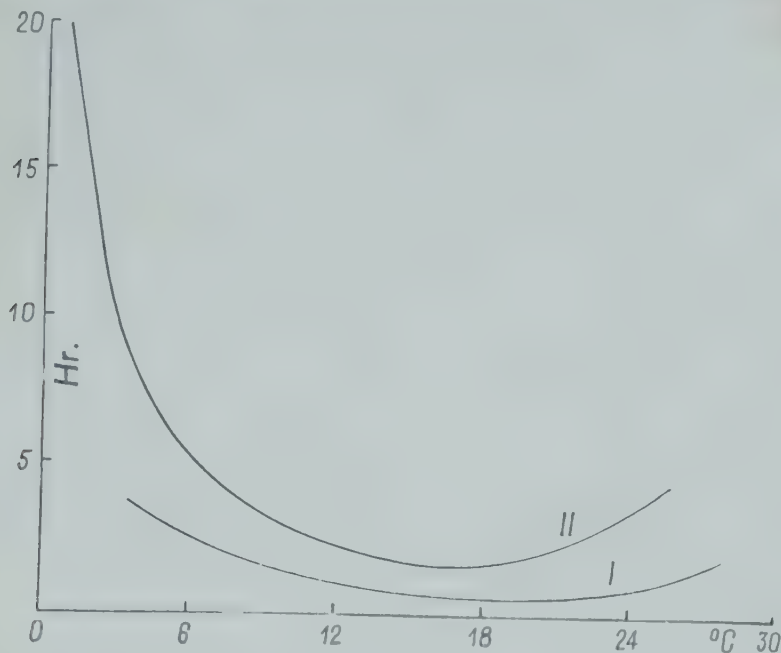


FIG. 29. The influence of air temperature on the speed of germination of conidia of *Pseudoperonospora humuli* (downy mildew of hop). Curve I: conidia harvested immediately after abstriction and at once set to germinate. Curve II: conidia which had been kept dry for 5 days after abstriction. Abscissae: temperature of germination. Ordinates: time required for swarmspore formation, in hours. (After Zattler, 1931.)

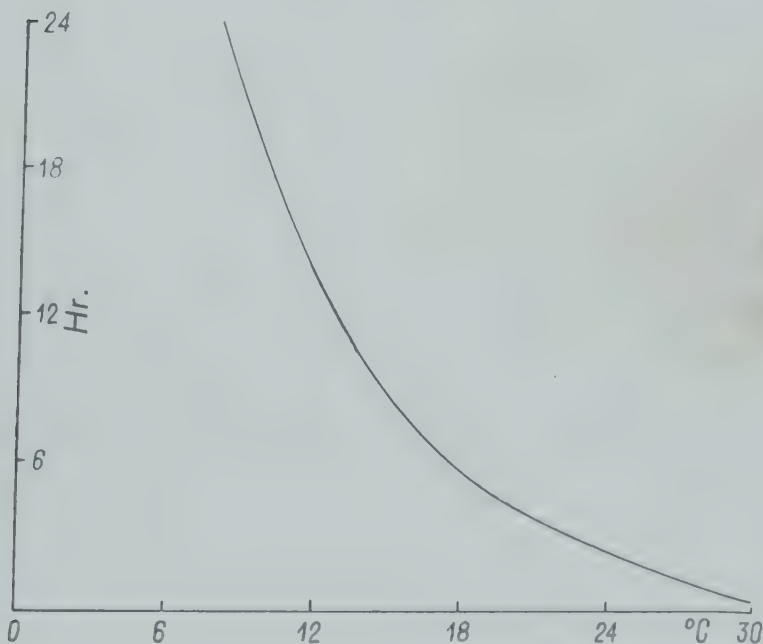


FIG. 30. The influence of air temperature on the duration of motility of the zoospores of *Pseudoperonospora humuli*. (After Zattler, 1931.)

middle of a hop garden on a warm summer's day; the parasite can, therefore, utilize nights of heavy dew but not close thundery weather. The cardinal points for infection are, on the whole, much the same as those

for germination, with the exception of the minimum which, for infection, is about 4° C.

Phytophthora infestans (potato blight) and *Plasmopara viticola* (downy mildew of vine) are relatively thermophilic with an optimum between

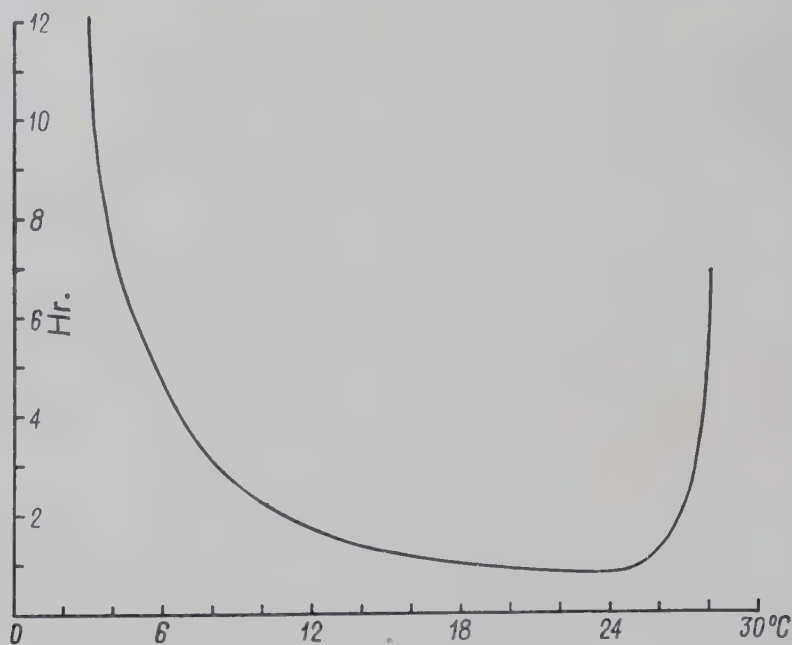


FIG. 31. The influence of air temperature on the speed of germination of the conidia (zoosporangia) of *Plasmopara viticola* (downy mildew of vine). (After Müller and Sleumer, 1934, and Maier, 1941.)

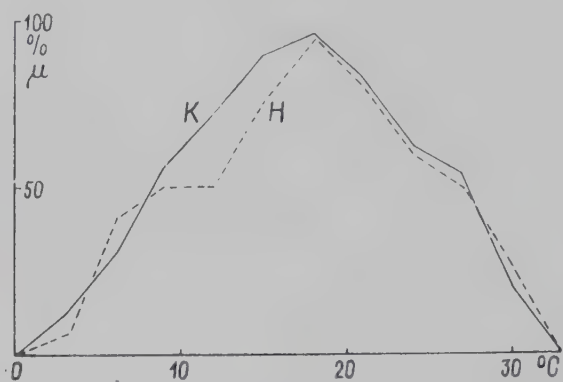


FIG. 32. The influence of air temperature on the germination of the conidia (curve K) and on the growth-rate of the germ tubes (length in μ after 5 days, curve H) of *Diplocarpon rosae* (black spot of rose). (After Frick, 1943.)

16 and 24° C., and the temperature curve (Fig. 31), after a sharp bend, reaches a maximum at 28° C. This, however, is only the upper limit for zoospore production; at 30° C. direct germination of the conidia with germ tube formation is still possible, a method of germination which, for all practical purposes, plays no part below 25° C. As in the case of downy mildew of hop, the zoospores of these fungi also remain viable for a longer period at low temperatures; in *Plasmopara viticola*, for example, they swarm for 20–28 hours at 6–7° C., for 10–16 hours at 15° C., for 4–10

hours at 20–22° C., for 2–3 hours at 27° C., and for only 15–60 minutes at 32° C. (Arens, 1929).

Again, the influence of external temperatures on the rate of growth of the germ tube varies, to some extent, with the species. In general, the cardinal points correspond to those for percentage germination (Fig. 32) but small discrepancies occur. Thus, the hyphal growth of *Clasterosporium carpophilum* (Fig. 25), in contrast to its percentage germination (Fig. 23), is very sensitive to temperature. Moreover, in *Puccinia dispersa* the optimum temperature for the growth of the uredospore germ tubes is 10–15° C. (showing some variation from the percentage rate curve, Fig. 27), and it is the same for *P. triticina*, while for *P. lolii* the optimum is 15–20° C. and for *P. graminis* it is 20° C. (Stock, 1931).

The three groups of factors here discussed, percentage germination, rapidity of germination, and growth-rate of the germ tubes, divergent as they are, represent only a small part of the temperature problem. It follows, therefore, that the influence of temperature on the result of infection cannot be predicted in advance but must be determined experimentally in each case. For example, in contrast to Fig. 27, Table III shows a pronounced optimum infection of black rust of wheat at an air temperature of 25° C.

TABLE III

The influence of air temperature on the intensity of infection by uredospores of Puccinia graminis tritici, biotype 3, on three species of Triticum. (After Peltier, 1923)

Host species	Average number of infection spots on the plumule at an air temperature of (°C.)			
	15°	20°	25°	30°
<i>Triticum vulgare</i> (Little Club)	5–10	5–25	25–50	1–10
<i>Triticum monococcum</i>	5–25	10–25	over 50	1–10
<i>Triticum dicoccum</i>	5–10	10–25	over 50	1–10

3. The Influence of Light on the Process of Infection

The influence of diffuse daylight (not direct sunlight) upon spore germination and upon the early stages of growth of the germ tubes is as variable as it is on the germination of the seeds of flowering plants. The oospores of *Plasmopara viticola* (downy mildew of vine; Arens, 1929), the conidia of *Colletotrichum Lindemuthianum* (anthracnose of bean; Lauritzen, 1919), and the uredospores of *Puccinia dispersa*, *P. triticina*, and *P. lolii* (Stock, 1931) germinate equally well in the light and in the dark, i.e. they are not sensitive to light.

The uredospores of *Puccinia graminis tritici* (black rust of wheat), on the other hand, are definitely checked by daylight (Dillon Weston, 1932) and germinate more abundantly and rapidly in darkness. In contrast, daylight stimulates the germination of the brandspores of bunt of wheat ('facultative

light germinators', Table IV), a fact that should be borne in mind when testing methods of plant protection.

TABLE IV

The influence of intensity of light on the germination of the brandspores of *Tilletia tritici* (bunt of wheat). (Hahne, 1925)

Exposure	very bright	bright	semi-dark	dark
days	%	%	%	%
3	4	0	0	0
5	61	30	0	0
12	92	81	10	8
17	92	84	35	20

4. The Influence of Hydrogen-ion Concentration on the Process of Infection

The infection droplet lying on a leaf surface is generally slightly alkaline (p. 6) and the optimum pH for germination might, therefore, be expected

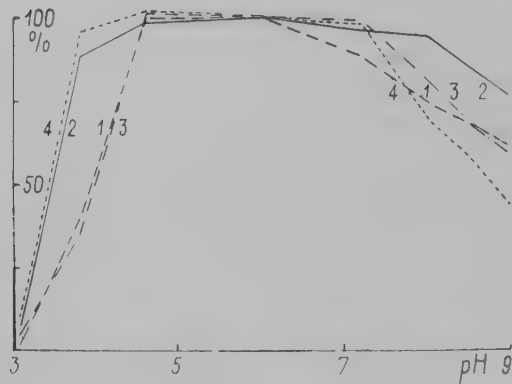


FIG. 33.

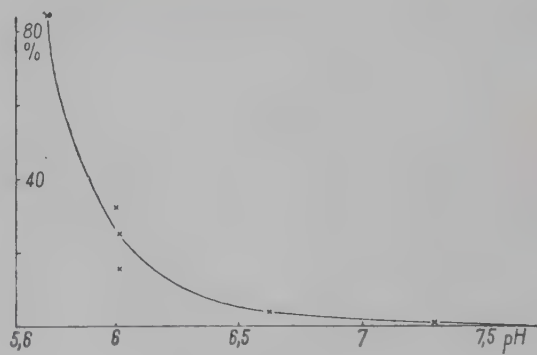


FIG. 34.

FIG. 33. The influence of the hydrogen-ion concentration of the substrate on the germination of the uredospores of some cereal rusts. Curve 1: *Puccinia triticina*. Curve 2: *P. dispersa*. Curve 3: *P. lolii avenae*. Curve 4: *P. graminis tritici*. (After Stock, 1931.)

FIG. 34. The influence of soil reaction on the occurrence of club root (*Plasmodiophora brassicae*). Abscissae: hydrogen-ion concentration. Ordinates: % of diseased plants. (After Chupp, 1928.)

to approximate to this value. The spores of parasitic fungi are, however, remarkably indifferent to the H- and OH-ions, in so far as they are allowed to germinate on natural substrates, properly buffered; the effect of ionization is closely dependent on the nature of the substrate. Fig. 33 shows that the optimum pH for the percentage germination of the uredospores of four cereal rusts lies between 4.6 and approximately 7.5, i.e. they are favoured by a medium with a slightly acid reaction.

Naked cells, such as myxamoebae or zoospores, are more sensitive to the acidity of the substrate than are cellulose-walled spores and germ tubes. Thus, the incidence of infection in club root (*Plasmodiophora brassicae*, Fig. 128) falls steeply from 100 to 20% with the slight change in pH from 5.7 to 6.2 (Fig. 34). A strongly alkaline soil reaction (pH 7.8) completely

inhibits the swarming of the myxamoebae, and therefore club root of crucifers can be controlled by heavily liming the soil; the myxamoebae die immediately on germination and infection is arrested.

§ 3. The Duration of Infection, Incubation, and Reproduction

Most phytopathological studies of the parasite-host complex overlook the time factor in relation to the infection, incubation, and reproduction periods.

The infection period is the measure of time required for the establishment of the parasitic relationship. It includes the period beginning with

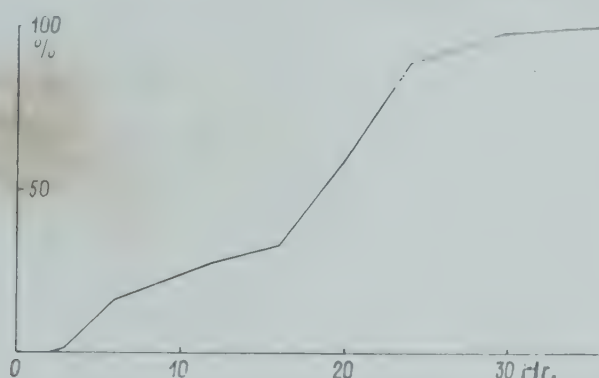


FIG. 35. The infection period of *Puccinia graminis tritici* (black rust of wheat) biotype 21, on the wheat variety Little Club at an air temperature of about 24° C. Explanation in text. (After Peltier, 1925.)

the germination of the parasite and ending in infection, i.e. until infection is so firmly established (p. 12) that neither changes in external conditions nor disinfection treatments will cause its breakdown.

As in the case of lethal temperatures (p. 22), the infection period represents not a point but a zone which allows for variation. Thus, in the early stages, when conditions affecting germination and development have only just been attained, only a few infections 'take', i.e. those produced by the most rapidly germinating spores, but as time passes the number of successful infections continually increases until, finally, an ideal incidence of 100% might be achieved. Fig. 35 represents graphically the results of spraying the leaves of a 7-day-old seedling with a uredospore suspension which is removed by drying at definite intervals (abscissae); the ordinates represent the percentage of plants infected. There is no infection when the suspension droplets are removed after a period of only 1 or 2 hours, 1.7% infection occurs when drying takes place 3 hours after spraying, 28% after 12 hours, 59% after 20 hours, 89% after 24 hours, and 100% after 36 hours. An uredospore of black rust of wheat can, therefore, not only germinate within 3 hours but also establish a relationship with the host cells so stable that it can no longer be destroyed by any change in the external conditions (incidence 1.7%); the minimum infection period, therefore, under the test conditions is 3 hours while, for 100% infection, 36 hours are necessary.

Similar minimal infection periods, about 2 hours, hold for the downy mildew of vine (*Plasmopara viticola*) and for potato blight (*Phytophthora infestans*). In loose smut of wheat which is referred to in greater detail later, actual infection is preceded by a complex transitional infection, so that infection first 'takes', at the earliest, 4 weeks after the brandspores have germinated.

These time data are valid only for the conditions present when they were estimated, which, in the above tests, were those for optimal germination and infection. On the other hand, as seen in Fig. 31, when, at an air temperature of 5° C. the conidia of *Plasmopara viticola* take 6 hours for germination alone, the total process of infection will not be completed in

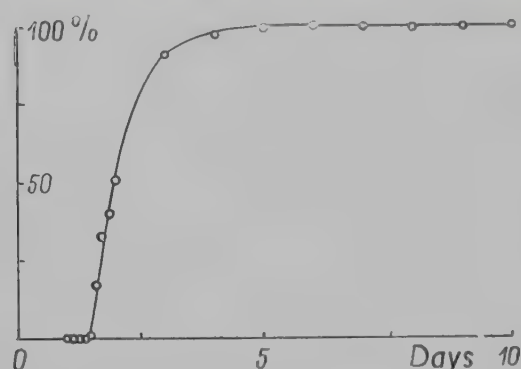


FIG. 36. The incubation period of tobacco mosaic virus in *Nicotiana glutinosa*, at an air temperature of about 24° C. Abscissae: number of days after inoculation. Ordinates: number of lesions. (After Holmes, 1932.)

less than 9 hours. The minimum infection period at an air temperature of 5° C. is, therefore, about 9 hours, i.e. some four times as long as at the optimum temperature.

The second time-span mentioned, the incubation period, is a measure of the progress of the parasitic relationship, with especial reference to the host. It includes the period from the commencement of infection, i.e. the beginning of the germination of the pathogen, to the crossing of the pathogenic threshold which, in turn, leads to definite external symptoms of

disease. During this symptom-free interval there take place in the host certain physiological changes which, later, become visible externally as the disease. Incubation is thus a time of preparation for disease, i.e. a hatching period. At the end of the incubation period the pathogen has established itself and the host begins to show definite signs of functional disturbance, i.e. the first clinical symptoms appear.

The length of the incubation period for each disease varies within certain definite and characteristic limits. In most parasitic plant diseases incubation lasts for about 10–14 days. For tobacco mosaic (Fig. 226) 1–2 days suffice, and for powdery mildew of cereals (*Erysiphe graminis tritici*) and others, from 5 to 7 days. On the other hand, leaf cast of pine takes 2–3 months to produce recognizable symptoms, whilst the parasite of witches' broom of fir (Fig. 230) takes about 4 months.

As a rule, the outward signs of disease which terminate the period of infection correspond not to a point but to a zone of variation representing the period during which the number of lesions (Fig. 36) and the severity of the disease symptoms are continually increasing; this stage may be regarded as the beginning of the disease proper.

In certain infectious plant diseases, more especially the mycoses, the concept of an 'incubation period' can be applied only with difficulty. In

medicine, where this term originated, incubation is often a matter of general disturbance and a state of being unwell, or there is a subjective response such as pain, or there may be a non-specific general reaction such as fever. Such possibilities are lacking in plant disease. For example, wheat plants affected by bunt or loose smut are infected throughout life and are diseased for months, showing increased respiration rate, disturbed protein metabolism, &c. But botanists, unlike medical men, cannot fall back on subjective symptoms such as the time when the plant 'begins to feel ill' and, therefore, they cannot determine precisely, e.g. in bunt or loose smut, the end of the incubation period and the beginning of the actual disease.

In such cases the phytopathologist is obliged to resort to the behaviour of the parasite, as in the third period mentioned, namely, the reproduction period.

The reproduction period, like the incubation period, is a measure of the progress of the parasitic relationship but this time with particular reference to the pathogen. It includes the time-span from the start of infection to the first appearance of the reproductive bodies of the pathogen, i.e. from the start of infection to the moment when both the host and the external conditions are favourable for the reproductive phase.¹ Thus, in bunt and loose smut, the reproduction period lasts from the germination of the brandspores and penetration of the germ tubes into the coleoptile or stigmatic papillae respectively, until the production of the brandspores in the ears of the host plant. It corresponds, therefore, with the entire developmental period in the host; infection taking place in the seedling or embryo stage, and reproduction at the time of flowering, i.e. of sexual maturity.

Parasitologically, the reproduction period is very different in character from the incubation period. During the latter the parasite is immersed within the tissues of the host whereas, at the beginning of fruiting, its reproductive organs usually emerge into the open and its spores are scattered abroad.

The time relations of the incubation and reproduction periods vary for the different infectious plant diseases. In a few exceptional cases the reproduction period is shorter than the incubation period. For example, under optimal conditions of temperature and humidity, the conidiophores of the downy mildew, *Peronospora brassicae*, break through the lower surfaces of the cotyledons of cabbage seedlings before any other definite symptoms of disease are apparent; these follow only later. The same is true for the downy mildew (*Peronospora cheiranthi*) of the wallflower (*Cheiranthus Cheiri*).

As a rule the parasite does not produce reproductive bodies until after incubation, i.e. until after the appearance of the disease symptoms, and the process occurs at definite periods of time characteristic for the different diseases. In the previously mentioned powdery mildew of cereals the change of phase from yellowing of the leaf tissues to the eruption of the oidia takes only one day under optimum external conditions. In the case

¹ [The 'reproduction period' is thus more correctly the 'pre-reproduction period' or the time to reproduction or to fructification.]

of the downy mildew of vine (*Plasmopara viticola*), there is, under optimum conditions, an interval of 1–2 days between the end of incubation, recognizable by the presence of the so-called oil spots, i.e. the chlorotic primary symptoms on the leaves, and the beginning of reproduction, when the conidiophores arise on the lower surfaces of the diseased leaves; this is the last possible moment for spraying the vines if the parasite is to be controlled. Also, in the case of the cereal rusts the reproduction period, which is ushered in by the appearance of the uredosori, begins only about 2 days after the end of the incubation period; the latter is terminated by the appearance of the chlorotic tissues which later become yellow and finally brown.

In certain other fungal diseases, however, the changes of phase occur at much longer intervals. Thus, in leaf cast of pine (*Lophodermium pinastri*), infection takes place at the end of May or the beginning of June; the characteristic yellow bands on the needles appear in September, i.e. the end of incubation; whilst the first fructifications, the pycnidia, are not seen until early in the following spring, i.e. about 6 months later.

In the case of witches' broom of fir (Fig. 230) the infection of the budding shoots by the rust takes place about the end of May. The first enlargement of the infected axial shoots, i.e. the end of incubation, shows itself in autumn; whilst the first reproductive bodies, the pycnidia and aecidia on the needles of the young brooms, only appear early in the following summer, i.e. about 9 months later. In the blister rusts of *Pinus* spp. (Fig. 52), the onset of the reproduction period may be delayed for as much as 2 years. In the case of the tree-inhabiting, wood-destroying Hymenomycetes the formation of brackets may be postponed even longer.

The relative length of the incubation and reproduction periods varies according to the susceptibility of the host and to external conditions, i.e. there may be a phenotypic displacement of the time of the appearance of the disease symptoms and of the reproductive bodies. These questions will be dealt with later in connexion with epidemiology (Chapter 2), with the influence of external conditions on the parasitic adaptation of the pathogen (Chapter 3), and with the disposition of the host as conditioned by environmental factors (Chapter 4).

§ 4. The Numerical Threshold of Infection

The density of the spore inoculum on the future host is termed the severity of infection or, somewhat ambiguously, the infection density; like the density of seed sowing in plant culture it may, in some pathogens, show a minimum, an optimum, and a maximum.

The minimum density is known as the numerical threshold of infection; it is the number of individual parasites which, under favourable conditions, is necessary to establish infection. This formally correct definition is of little practical use as the focus of infection has to be destroyed in the process of determining microscopically whether infection has 'taken'. As a rule,

therefore, the pathologist establishes the occurrence not of infection itself but of its successful outcome; i.e. he ascertains how many individual parasites are required to produce disease under favourable circumstances. Thus, the spot where an organism has become diseased indicates where infection has 'taken'. However, not every infection need bring about disease; to avoid mistakes, therefore, it is necessary to make sure to which of these two threshold values, that of infection or that of disease, a writer refers.

In human and veterinary medicine a single bacterium of anthrax, for example, will cause death in mice in 28%, cases of the disease whereas twenty germs are 100% lethal to mice. It is the same for the pneumococci of pneumonia in rabbits and for tubercle bacilli in guinea-pigs; on the other hand, goats must inhale 0.01 mg. of tubercle bacilli in order to develop tuberculosis.

The value of the numerical threshold depends not only on the infective ability of the parasite but also on the resistance of the host; in susceptible hosts the probability of infection by the three bacteria mentioned is extraordinarily high and, correspondingly, the numerical threshold values are very low. On the other hand, with moderately virulent typhoid bacilli, about 30,000 individuals are required to produce one death in 80% treated mice, while several milligrammes of meningococcal culture, the causal organism of meningitis or 'brain fever', are required for a dose lethal to mice (Zinsser *et al.*, 1939); in these two pathogens the capacity for infection is slight and the numerical threshold value is correspondingly very high.

Similar relationships hold good in plant pathology. Here also there is a first group of infectious diseases in which a single spore will suffice to produce an infection, for example, an uredospore of a rust fungus or an oidium of a powdery mildew (*Erysiphaceae*) can produce a normal infection on a highly susceptible host. It is this fact that makes it possible in these obligate parasites, i.e. parasites which cannot be grown on an artificial medium, to obtain single spore cultures and thus separate the various biotypes and strains.

In virus diseases, also, the minimal dose may be very low. In tobacco mosaic, 10^{-13} g. protein (i.e. about 300 molecules) per c.c. is effective; this corresponds to a dilution of the virus-containing cell sap of about 1:10⁶. In the *N*-strain of the potato virus X-group, on the other hand, the lower limits of dilution of the virus-containing cell sap is 1:10⁵, in the *G*- and *S*-strains 1:10⁴, and in the *H*-strain 1:3,000 (Salaman, 1938).

In this first group of pathogens a single spore or a single virus molecule can lead to infection, but only under optimal conditions; hence, with very low numbers the probability of success is slight.

In a second group of plant diseases a single spore, even under optimal conditions, will not give rise to an infection; in these cases a mass infection is required to ensure 'taking'.

The numerical infection thresholds in the several diseases of this group are varyingly high. In wart disease of potato (Fig. 37) 200 resting sporangia per gramme of soil are necessary to cause disease; in some bacterial diseases

(Fig. 60) a whole zoogloea must be present; whilst in snow mould of wheat (Fig. 38) not less than 10,000 conidia per c.c. of inoculum are needed. In tomato wilt even a virulent strain (Fig. 39, curve *A*) requires 700,000 conidia per c.c. of inoculum to bring about disease. The bunt-susceptible

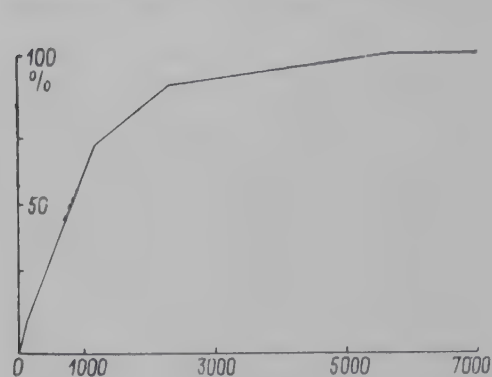


FIG. 37.

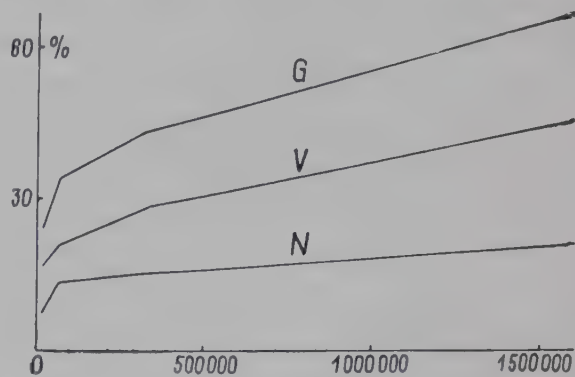


FIG. 38.

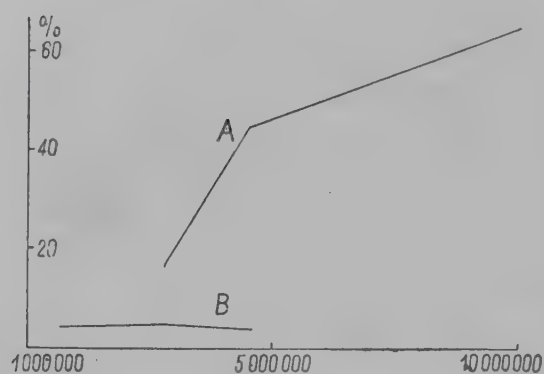


FIG. 39.

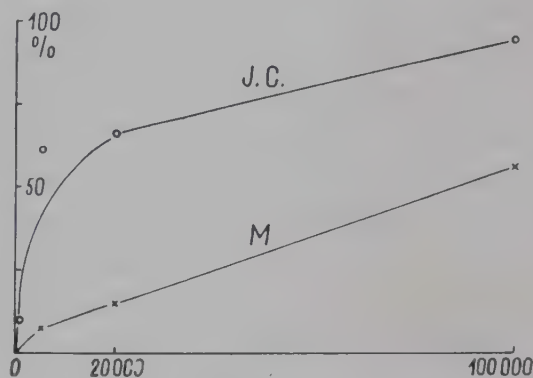


FIG. 40.

FIG. 37. The influence of inoculum density on the incidence of wart disease of potato (*Synchytrium endobioticum*). Abscissae: approximate number of resting spores per g. of soil. Ordinates: number of diseased plants. (After Glynne, 1925.)

FIG. 38. The influence of inoculum density on the incidence of snow mould disease of wheat seedlings (*Gibberella Saubinetii*). Abscissae: number of conidia per c.c. of inoculum suspension, in which the grains were immersed for 10 minutes before sowing. Ordinates: number of seedlings badly diseased before surfacing (curve *V*) and after surfacing (curve *N*). Curve *G*: total number of seedlings affected. (After Dickson, 1923.)

FIG. 39. The influence of inoculum density on the incidence of tomato wilt (*Fusarium lycopersici*). Abscissae: number of conidia per c.c. of inoculum suspension sprayed on the soil after the seedlings had surfaced. Ordinates: number of diseased seedlings resulting from attack by a strongly aggressive strain *A*, and by a weak strain *B*, respectively. (After Haymaker, 1928.)

FIG. 40. The influence of inoculum density on the incidence of loose smut of wheat (*Tilletia tritici*). Abscissae: approximate number of brandspores per grain of wheat. Ordinates: number of diseased plants of the susceptible variety Jenkins Club (curve *J.C.*) and of the resistant Marquis wheat (curve *M*). (After Heald, 1921.)

Club variety of wheat (Fig. 40) becomes diseased if attacked by 100 brandspores per wheat grain, whilst the resistant Marquis variety needs 500–5,000 brandspores.

At least four groups of factors interact to determine the actual level of infection in these pathogens:

1. The probability of attack. This is the statistical probability that the

spores of the parasite will germinate at the right moment and will reach, again at the right moment, a susceptible area of the plant to be attacked. These conditions are clearly present in all the four examples given, see especially Figs. 38-40; in these every particle of soil and every germinating grain of corn is abundantly supplied with spores, so that numerous germ tubes are able to contact each host plant.

2. The spatial relations of germination. If the infection droplet or the amount of solution be too great in comparison with the number of spores, the enzymes and growth substances diffusing out of the germ tubes will become too dilute and the pathogen will starve. Conversely, if the spore density in the infection droplet be increased, then their concentration is increased in geometrical progression. As shown in Fig. 41 *Ustilago violacea* cannot develop in a dilute suspension without the addition of growth substance, whereas in a concentrated inoculum it develops well.

3. The pathogenic efficiency of individual parasites. An infection spot due to one myxamoebe of *Synchytrium endobioticum* does not lead to wart disease (Fig. 187); the morphogenic stimulus is too weak. For disease to ensue, i.e. for the production of obvious signs of functional disturbance in the host, a large number of infecting organisms or even a mass attack by the parasite is required. Again, in the case of the two *Fusaria* (Figs. 38 and 39) a single infection spot, i.e. one penetrating hypha, is ineffective; its pathogenic efficiency is not great enough. In these parasites the incidence of disease is, consequently, much lower than the incidence of infection.

4. The resistance of the host. Fig. 40 depicts two varieties of wheat which have been dusted with an equal quantity of spores at the same time and under the same external conditions; the statistical probability of attack including the incidence of attack in the narrower sense of the word (the number of germ tubes which penetrate, i.e. the localized infection mass) is, therefore, probably equal in both cases. Yet, for disease to occur in the susceptible Club variety only 100 brandspores are required, whereas the resistant Marquis variety needs approximately ten times as many.

Since the importance of these four factor-groups varies with the external conditions, any generally valid rules regarding the location of the numerical infection threshold are scarcely possible in connexion with the pathogens under discussion.

If, in either group of pathogens, the amount of inoculum be increased

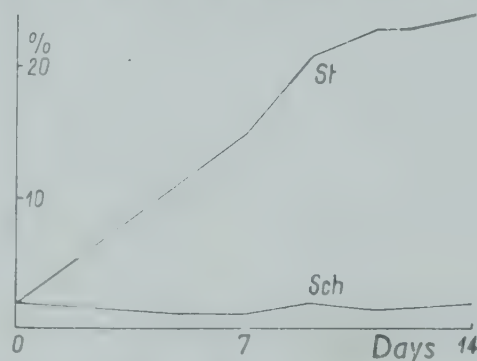


FIG. 41. The influence of inoculum density on the growth of *Ustilago violacea* (anther smut of Caryophyllaceae) in synthetic media without growth substance. Curve *Sch*: weak inoculum (one drop of the spore suspension per test tube). Curve *St*: inoculum 200 times as strong. Abscissae: time in days. Ordinates: growth intensity measured according to nephelometric turbidity. (After Schopfer and Blumer, 1938.)

above the numerical threshold needed for infection, the probability that infection will 'take' and, with it, the incidence of disease, will increase to an optimum. This is shown in Fig. 42 where the virus-containing sap diluted to 1:1,000 gave 45 lesions per leaf, to 1:100 gave 230, to 1:10 about 660, and to 1:1 gave 1,620. (Mathematical analysis given by Youden *et al.*, 1935.)

The optimum infection dose gives rise to the maximum amount of disease that the particular parasite can produce on the particular host under the given conditions. This maximum incidence of disease will vary with the parasitic adaptation of the pathogen and with the disease proneness

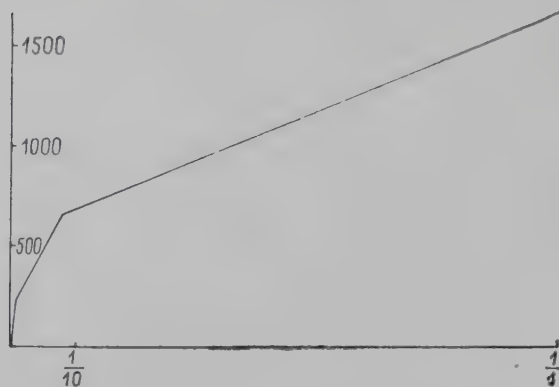


FIG. 42.

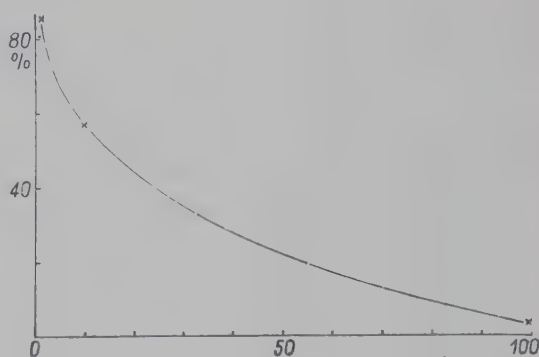


FIG. 43.

FIG. 42. The correlation between the number of lesions (single spots) and the concentration of infection material. Abscissae: degree of dilution of expressed sap. Ordinates: average number of lesions caused by tobacco mosaic virus on leaves of bean (*Phaseolus vulgaris*). (After Price, 1930.)

FIG. 43. The influence of inoculum density on the infection of the petals of sweet pea (*Lathyrus odoratus*) with conidia of *Botrytis cinerea*. Abscissae: relative density of spore suspension. Ordinates: % of successful infections. (After Brown, 1922.)

of the host. In Figs. 37 and 42, 100% of the individuals are infected, whereas in Fig. 38 and the Marquis wheat in Fig. 40, the maximum is 60%. The highest values give the maximum amount of disease which can be produced by the activity of the particular parasite but not the optimal use of the infection material. All the curves in the illustrations referred to are asymptotic, and it thus requires disproportionately more infective material to raise the disease incidence from 90 to 100%.

In some infectious diseases, e.g. those viroses caused by potato virus X (Fig. 148) and tobacco ring spot virus (Fig. 223), an increase in the quantity of inoculum only raises the probability that infection will 'take'; the severity of the disease is not increased since it is not dependent upon the amount of inoculum. In these cases, once infection has 'taken', the disease runs its course with the degree of intensity characteristic of that particular parasite-host relationship.

Usually, however, by increasing the quantity of inoculum not only the incidence but also the severity of the disease is increased. This mass action is due to the localized effect of the cell wall dissolving enzymes secreted from numerous hyphae (enzyme activity increases about 1.5 times

with every unit of increase in enzyme concentration) and to the additive effect of their toxic secretions which exceed the local threshold value and induce a local breakdown of host resistance.

It is only in rare cases that an inoculum increased in quantity above the optimum has no effect; for instance, in the viroses mentioned above the incidence of the disease remains optimal. In general, the result of infection is diminished by increasing the concentration of the inoculum above the optimum (Fig. 43).

This effect may be due to a deficiency of oxygen, to mutual inhibition by metabolic products, and in many cases simply to competition for nutrients. The steep fall of the curve in Fig. 43 applies only to such substrates as the petals of *Lathyrus odoratus*, which have weak powers of exosmosis, and not to the same extent to the freely excreting petals of *Cereus spectabilis* (Fig. 2).

Where there is an excessive infection dose the competition for nutrients does not stop with the entrance of the parasite into the host but goes on inside the host tissues. In wart disease of potato (Fig. 44) the majority of the invading parasites in a too dense spore suspension die. The pathogen utilizes not only the material of the host cells which it directly invades but also substances which diffuse from the neighbouring cells. If these cells have already been colonized then the available nutrients are insufficient to support all the spores.

§ 5. The Path of Infection

The following section is concerned with the entry of the parasite into the host; the means by which it reaches the host will be discussed in the next chapter ('Epidemiology'). The most important modes of entry of the parasite into the host are as follows:

- (a) by attacking uninjured parts of the plant,
 1. the intact surface (p. 44);
 2. the natural openings (stomata, lenticels, &c.; p. 44);
 3. special organs (stigma, root-hairs, &c.; p. 49);
- (b) by attacking damaged areas,
 4. wounds due to mechanical or physical causes (p. 54);
 5. wounds caused by other parasites (p. 56);
 6. wounds produced by the vectors of the pathogens (p. 57).



FIG. 44. The adverse effects on the invading pathogen due to an excessive inoculum density. A young leaf of the potato variety Wohltmann invaded by too many myxamoebae of *Synchytrium endobioticum* (wart disease of potato). When the photograph was taken, 14 days after inoculation, the majority of them (indicated by the arrows) were already dying. Left, a large immature resting sporangium. $\times 310$. (After Köhler, 1931.)

The modes of entry included under (a) are termed 'natural' and those under (b) 'artificial'. A few examples from each of these six groups are described briefly below.

1. Entry through Intact Surfaces

This way of infection is often used by plant pathogenic fungi (p. 6). A typical example is the active penetration of *Botrytis cinerea* through the cuticle, &c., into the interior of leaves; this fungus can also penetrate, by a similar process, into organs which are covered by protective layers other

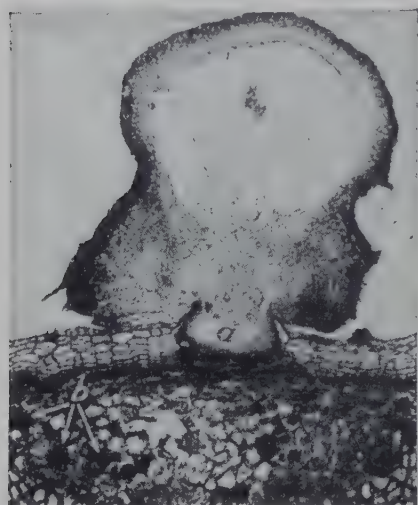


FIG. 45. A mycelial strand of the honey agaric (*Armillaria mellea*) penetrating into an uninjured root of walnut (*Juglans regia*). *a* invading strand, *b* reaction zone. $\times 41$. (After Thomas, 1934.)

than cuticle. Similarly, the rapidly growing strands or rhizomorphs of the honey agaric (*Armillaria mellea*), which are found growing in the soil, can enter directly through wounds in the roots or base of the trunk of deciduous and evergreen trees, but, in the absence of a wound, the rhizomorphs can attach themselves firmly to an undamaged root by means of a slimy substance which is found surrounding the area immediately behind their white growing tips. Some hyphae grow out from the tip of the strand into the superficial layers of the root cortex and by this means the strand becomes even more firmly attached to the root. At the point of contact the strand forms a branch which, by a mechanical action similar to that used by the haustoria of the mistletoe (Fig. 10), breaks through the epidermis and

periderm simply by pressing on the host cells (Fig. 45, *a*). Infection of undamaged tree roots by *Armillaria mellea* is achieved not by single hyphae but by mass attack of the strands themselves.

Following the penetration of the cork layer, the toxic substances produced by the hyphal strand become effective. As a reaction to this the cells of the cortical parenchyma at some distance from the entering hyphal strand undergo changes; their contents become plasmolysed and darken in colour, similarly the cell walls and nuclei become discoloured, and finally the cells die and become filled with wound gum (Fig. 45, *b*). In susceptible hosts the invading strand then forms another series of side branches which penetrate laterally into those parts of the cortex and cambium which have already been killed by toxins and, in a short time, bring about the effective poisoning and disease of the tree.

2. Entry through Natural Openings

The natural openings, stomata, lenticels, &c., serve as points of entry for plant parasites just as frequently as do the intact surfaces. On the whole, however, they are of less importance than in human beings, whose mouth

and nose are primary avenues of infection. This is partly because of the peculiarity of our breathing process in which we suck the air actively into the respiratory organs and thus introduce the pathogens contained in it, i.e. there is direct inhalation of pathogenic germs, and partly because of the peculiarity of our method of feeding, the mouth forming an entrance to the digestive organs and allowing the germs, which are invariably present in food and drink, to enter into the body.

In both these respects, the natural openings of plants, especially the stomata, are of minor significance. Although the stomata are exceedingly

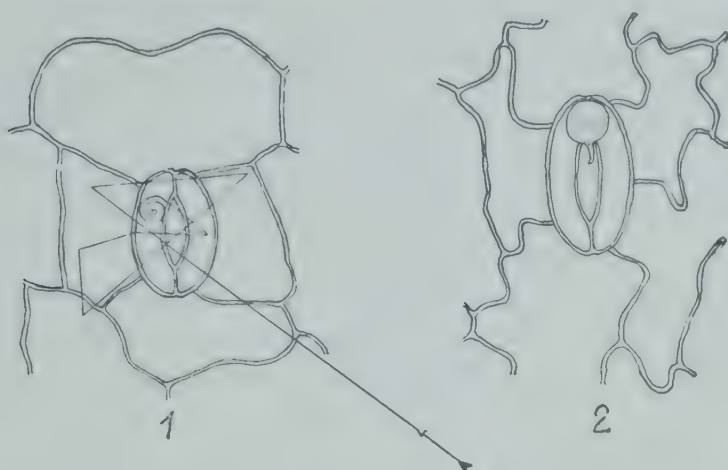


FIG. 46. The movement of zoospores of *Plasmopara viticola* (downy mildew of vine) through the stomata of a vine leaf. 1 Path of the zoospore in the infection drop after it has reached the neighbourhood of a stoma. 2 Zoospore which has settled down on the mouth of a stoma and germinated. Approx. $\times 1,000$. (After Arens, 1929.)

numerous, approximately 5,000–20,000 per sq. cm. of leaf surface, they are very small, approximately $10\ \mu$ long and, when wide open in the light, $0.5\text{--}0.3\ \mu$ wide, so that they exclude most spores by mechanical filtration. In addition, respiration in plants is passive; it is not a process of alternating inspiration and expiration of air but of quiet physical diffusion conditioned by diffusion gradients, and the intake of germs is, therefore, scarcely probable. Further, in the plant there is no link between infection and feeding as the latter takes place entirely by osmosis through the cell walls.

The mechanism of penetration through the natural openings varies according to the kind of parasite involved. The entry of plant pathogenic bacteria through the stomata takes place passively as they are drawn into the pores in drops of water (p. 17). In the case of bacteria, lenticels hardly need to be considered because of their suberization. On the other hand, the germ tubes of plant pathogenic fungi actively push down through the mouths of the stomata or lenticels in response to a chemotropic stimulus, in a way similar to that in which they penetrate the cuticle, because the stomatal openings are not wide enough for them. They also force themselves down through narrow fissures as is shown by the constriction of the infecting hypha in Fig. 1. Hence, stomatal infections also occur at night because 'closed' stomata are never completely shut.

The zoospores of *Plasmopara viticola* (downy mildew of vine, Fig. 26) at first swim around in the infection drop by means of their flagella, or slide about on the epidermis on their grooved sides; when they come near to a stoma they begin to react and show 'purposive' tactical movements towards the stomatal opening (Fig. 46, 1). Thus, they 'seek' the place of infection actively, and when they reach it they settle down upon it, round themselves off, and surround themselves with a membrane (Fig. 46, 2); after only 12 minutes, under favourable conditions, a germ tube is driven down into the stomatal chamber.

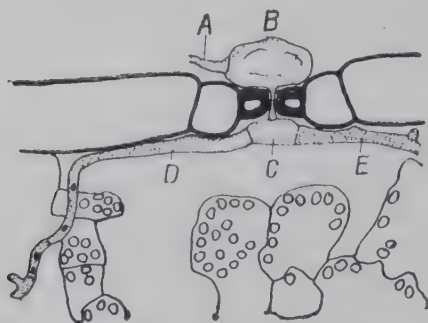


FIG. 47. The penetration of the germ tube of *Puccinia coronata* into a stoma of *Avena sativa*. A residual germ tube, B residual appressorium, C empty sub-stomatal vesicle, D and E infection hyphae penetrating the mesophyll. $\times 360$. (After Ruttle and Fraser, 1927.)

A similar chemotropic response is found in all the fungi which germinate directly by means of a germ tube. Thus, the germ tubes of the uredospores of crown rust of oats (*Puccinia coronata*) at first appear to grow aimlessly over the surface of the leaf. When they reach a stoma they are obviously stimulated by the gaseous products; the protoplasm accumulates at the tip which swells into an appressorium over the stomatal pore (Fig. 47, B). The nucleus divides and, consequently, the appressorium contains usually four or more nuclei. Later,

a fine process penetrates between the guard cells into the air space below and there it swells again into a vesicle C, the sub-stomatal vesicle, which usually possesses eight nuclei. Branch hyphae, the true infection hyphae D and E grow out from this vesicle, branch in all directions, and enter the intercellular spaces. In this way the germ tubes of many spores can use the same stoma.

The same effect is shown in Fig. 48. Two germ tubes K of *Lophodermium pinastri* (leaf cast, or red leaf of pine) have entered a stomatal pit V and each has there swollen into a vesicle. This has formed side branches which have attacked the guard cells SZ and put them out of action; they have then grown laterally into the hypodermis H, and have disorganized the parts of the epidermis E lying between them (DE). In the air space A they again branch and have penetrated the intercellular spaces of the mesophyll M, where they have dissolved the middle lamella and finally the cell walls (the decomposition of the mesophyll AM is indicated by dotted lines for the cell walls). The endodermis ring EN can be broken through only by penetration through a middle lamella; there is a marked thickening in the walls of the adjoining endodermal cells which is obviously a wound reaction. Subsequently, the fungus has grown by the shortest route through the thin-walled elements of the transfusion tissue T into the vascular bundle where it has grown through the phloem P, into the medullary rays MS and the wood parenchyma XP; the true vessels and tracheids are, however, avoided.

In other needle-leaf diseases, on the other hand, the method of stomatal infection is more complex. For instance, the infection hyphae of *Herpotrichia nigra* (Fig. 71, brown felt blight), of the Swiss mountain pine (*Pinus montana*), and of *Phaeocryptopus Gäumanni* (syn. *Adelopus Gäumanni*), which in Switzerland causes needle cast of Douglas fir, begin by forming a coiled mycelial plug in the entrance pit of the stoma. At first, this serves as the starting-point for the true endoparasitic colonization of the host which, in *Herpotrichia*, takes place laterally through the epidermal and hypodermal cells thus circumventing the stomatal chamber (Gäumann *et al.*, 1934). In *Phaeocryptopus* it takes place directly through the chamber, and the mycelial plug later grows into the primordium of a fructification (Fig. 49).

Whether the parasite uses the natural openings as an entry point or perforates the cuticle depends, in certain cases, on the parasite. Thus, the germ tubes of the basidiospores of most rust fungi 'ignore' nearby stomata and penetrate directly through the cuticle. This is also the case in many species of the powdery mildews (Erysiphaceae, Fig. 76), in *Pseudopeziza tracheiphila* (Rot-brenners of vine), and in the cuticular invaders such as *Endostigme inaequalis* (apple scab, Fig. 73) and *Diplocarpon rosae* (black spot of rose, Fig. 75). In these fungi the germ tubes have to bore their way through the cuticle because clearly they are unable to make use of the stomata.

Conversely, other fungi are entirely dependent on natural openings because they are unable to penetrate the thickened superficial layers mechanically and must, therefore, enter where they can. For instance, the germ tubes of the uredospores of most rusts are not able to pierce even lightly cutinized layers and, in consequence, only enter the stomatal side of the leaf (Fig. 47) although, if the cuticle be damaged, they can also infect the leaf from the upper surface. In a similar way, *Spongospora subterranea* (powdery scab of potato) and *Actinomyces scabies* (common scab, Fig. 50) are obliged to use the lenticels because they cannot break through the

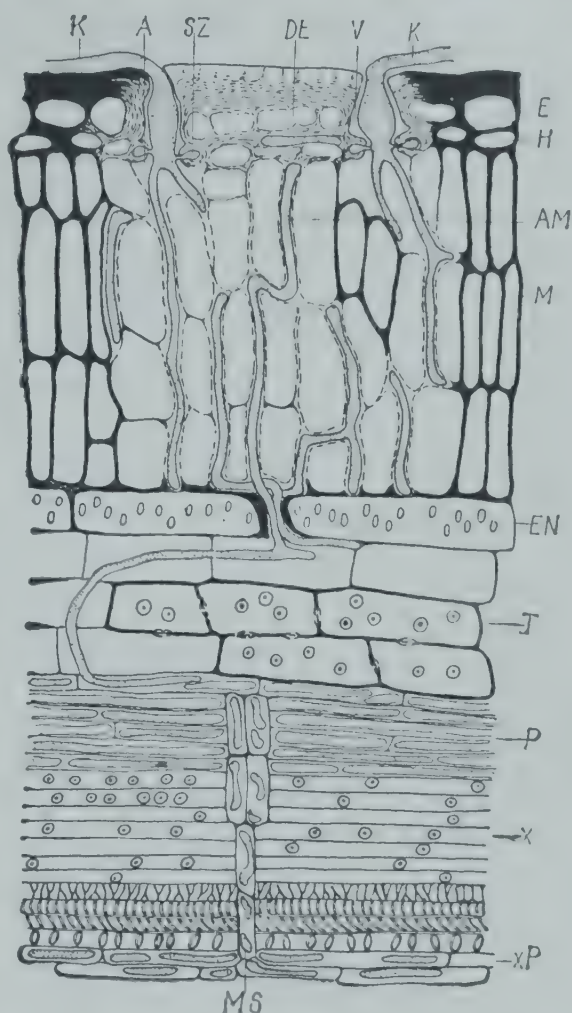


FIG. 48. Attack on a pine needle by *Lophodermium pinastri*. Explanation in text. Approx. $\times 750$. (After S. G. Jones, 1935.)

periderm. Similarly, the germ tubes of *Lophodermium* (Fig. 48) must enter through the stomata as they cannot perforate the cuticle, which may be as much as $4\ \mu$ in thickness. With these fungi there is no question of a method of infection like that of the powdery mildews (Fig. 76).

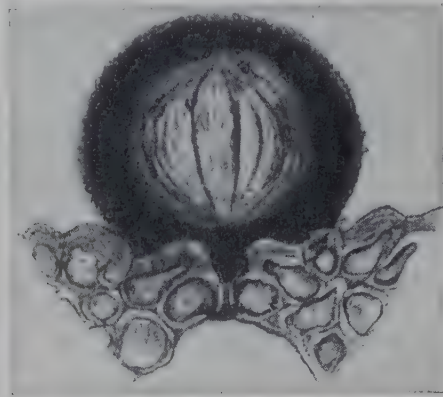


FIG. 49. Section through a pseudothecium of *Phaeocryptopus Gäumannii* which has developed from an infection plug. The asci are immature. Approx. $\times 400$. (After Rohde, 1937.)

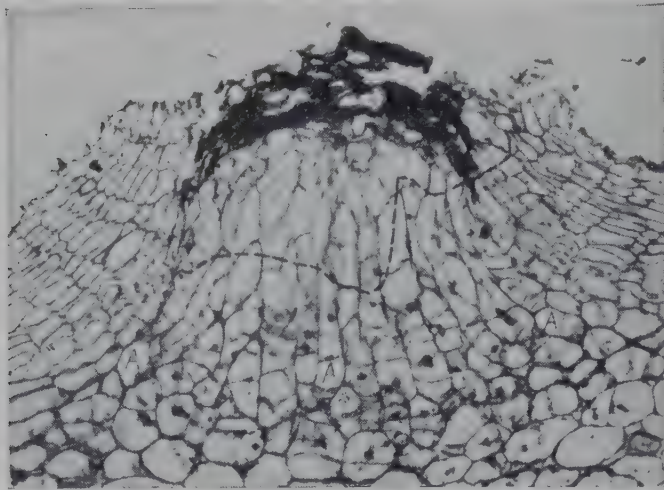


FIG. 50. Lenticel infection in potato scab. A young lenticel infected (as far as the dotted line) by *Actinomyces scabies*; the peripheral parts are already dead. A-A lenticel meristem. $\times 65$. (After Jones, 1931.)

Finally, still other pathogens are not particular and use any method. Thus, the germ tubes of the zoospores of *Phytophthora infestans* attack the potato plant through the cuticle as well as through the stomata and the tubers through the lenticels as well as through wounds. *Sclerotinia cinerea* (brown rot of unripe stone fruits), *Botrytis cinerea* (grey mould of stored apples), and *Nectria cinnabarina*, the agent of coral spot disease of street trees, invade their hosts, depending on the circumstances, either through the lenticels, through the cuticle (if it be weak), or through wounds.

Sometimes the infection path is conditioned by the state of health of the host. Thus, *Botrytis cinerea* can infect healthy leaves of *Vicia Faba* only

through the cuticle (Fig. 1), but if the leaves are already diseased the germ tubes can make their way through the stomata.

3. *Entry through Special Organs*

Some parasites of infectious plant diseases use certain organs, without injuring them, merely as entrances or passages into the undamaged host. They grow through these organs towards entirely different organs in which they give rise to the characteristic disease and in which they may reproduce. The importance of these specialized avenues of infection was first recognized in human medicine; thus, cholera and typhoid bacteria are not able to establish themselves directly through the skin but only in the mucous membrane of the digestive tract, from whence, particularly in typhoid, they make their way into the circulatory system.

In particular, the flowers, seedlings, buds, needle leaves, and root-hairs serve as such 'Trojan' entry points for infectious plant diseases.

(a) *Flower Infection*

In flower infection the stigma, because of its secretions and special structure, offers the parasitic micro-organism the most favourable substrate for its germination and tissue for its initial growth. Such stigmatic infection is very often found in *Sclerotinia cinerea* (syn. *Monilia cinerea*), which causes the well-known blossom and twig blight of stone fruit trees and the pustules on infected fruits, especially the cherry, and in *Sclerotinia Linhartiana*, which causes a similar disease of the quince. The conidia germinate, like the pollen grains, on the stigmatic papillae and the germ tubes penetrate through the style into the ovary just as do the pollen tubes. From here the hyphae destroy the whole flower and then grow through the peduncle into the branch below where, after 3-4 weeks, they produce a sudden wilt of the whole branch and leaves (M. Schmidt, 1938).

Small injuries, cracks in the skin, frost damage to buds, &c., can also serve as points of entry. Thus, in early infections the stigma is only the most usual and not the exclusive path of entry, the *locus minoris resistentiae*. Late infection of fruits, on the other hand, takes place directly through lenticels, &c. (p. 47).

In addition to the stigma, flower infection also takes place, although rarely, through the nectaries. These have no cutinized layer, and like the stigma their sugary secretion provides a favourable medium for micro-organisms. This path of infection is used, e.g. by *Bacillus amylovorus* (fire blight, Figs. 15 and 16). The bacteria are transferred to the nectary by bees during pollination; from here they invade the ovaries, preventing their development, and then attack the twigs and branches. In midsummer the dead leaves hang down browned or blackened as though they had been singed, hence the name of the disease. Finally, the bacteria pass into the trunk and roots and destroy the whole tree. Bacteria can also be introduced into foliage leaves by the proboscides of sap-sucking insects or through

wounds in the branches and, similarly, originate a progressive disease of the whole tree.

Attack through the flower, i.e. through the reproductive organs, is not, therefore, in either fire blight or *Sclerotinia* twig blight, the only possible way of infection but it is the commonest and most effective way. A parallel type of infection is found in the venereal diseases of man.

The flower infections mentioned above are of the nature of direct infections. The reproductive organs form the specific and natural path of infection into the plant to which they belong, which thus becomes diseased. On the other hand, in another type of flower infection the reproductive organs of the mother plant serve only for the reception and spread of the disease germs; the mother plant whose flowers receive the germs does not itself become diseased but transmits the germs to its progeny without itself showing any symptoms. It is only the daughter plants that become diseased.

These indirect infections will be considered in the next chapter ('Epidemiology'). They include, on the one hand, transitional infections, e.g. flower-seedling infection, as in loose smut of oats and stripe disease of barley, and, on the other hand, the germinative transmission of infection, for example, from the mother plant, as in the loose smuts of wheat and barley. In both of these it is not a matter of finding the way into a host but of finding the way to a specific host.

(b) *Seedling Infection*

In the second group of specific avenues of infection, seedling infection, the seedling is infected but, in contrast to the true seedling diseases, it does not itself become diseased; it harbours the germs without showing any symptoms. The seedling grows into a plant in the normal way and the parasite develops simultaneously in its tissues. The disease first appears some months later, often at the time of flowering, in an entirely different part of the plant such as the female or male organs or the corolla.

Such seedling infection causing disease in the female organs occurs in bunt of wheat. The brandspores of the pathogen, *Tilletia tritici*, stick to the beard of the wheat grain and reach the soil with the seed, or they may already be in the soil. They germinate at the same time as the grain and the hyphae penetrate into the seedling in the normal way. Subsequently, the hyphae penetrate intercellularly to the growing-point, and develop just behind it throughout the vegetative period. From the growing-point the fungus invades the young leaves and produces a series of mild, non-specific, general symptoms in the infected plant. These symptoms will be discussed later (Chapter 5: 'The Disease').

Finally, the hyphae attack the flower primordia, invade the seed, use up the reserves of the young endosperm, destroy the embryo, and then form spores; the bunt ball consists of an evil-smelling spore mass surrounded by the pericarp (stinking smut). In this case, therefore, infection takes place in the seedling whereas the disease breaks out in the ear, or rather in the seed.

If, owing to favourable growing conditions, the infected plant develops especially quickly, its growing-point out-distances the parasite, leaving it behind in the culm and leaves, and the disease remains latent, i.e. it does not break out at all.

Seedling infection leading to the outbreak of disease in the male reproductive organs occurs in the anther smuts of Caryophyllaceae (*Ustilago violacea*). As in bunt of wheat, infection takes place normally through the seedling but shoot infection can also occur, entrance being made through the young tissues and buds of the growing shoots. As in bunt, the hyphae develop just behind the growing-point until flowering time and spread into the developing branches and leaves. They give rise in the infected plant to a series of non-specific, general symptoms, e.g. retardation of growth and of root formation, yellowish-green colour due to disturbance of the chlorophyll apparatus, increased stooling and hairiness, and arrest of differentiation in the leaf tissue so that in extreme cases palisade and spongy mesophyll are indistinguishable (Blumer, 1941).

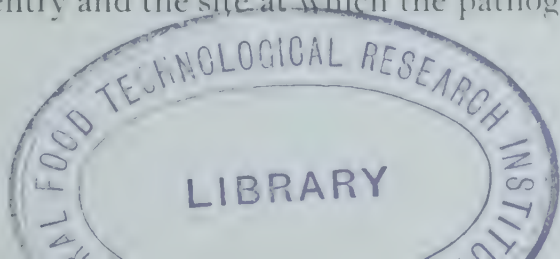
Finally, the mycelium invades the anthers and, in contrast to bunt, produces its fruiting bodies and attains its full pathological effects in the male organs. The latter become sterile because the formation of pollen is prevented and, instead, the parasite forms its smut spores in the pollen chambers. The outer cells of the anther at first remain intact but, later, the brownish-violet spore masses become free and are scattered by their dehiscence. The mycelium perennates within the infected plant so that destruction of anthers occurs repeatedly every year.

Lastly, seedling infection with the outbreak of the disease in the corolla presumably occurs in some downy mildews especially *Peronospora Radii*, which is found in the corolla of several Compositae (*Achillea*, *Anthemis*, *Chrysanthemum*, and *Matricaria*), and in *Peronospora corollae*, on the corolla of *Campanula persicifolia* and *Linaria vulgaris* (Gäumann, 1923).

Evidently the pathogen enters the seedling in spring from overwintering oospores (resting spores), grows with the growing-point until flower formation without producing any symptoms, and then reproduces in the corolla. It is only here that the disease first breaks out, i.e. not in the male organs, as in the anther smuts, but in their derivatives.

(c) Bud Infection

In the third group of specific paths of infection, bud infection, disease ensues only when infection takes place sufficiently early, as in the previous case of seedling infection, to ensure that the causal agent reaches the growing-point. The parasite can pass through other tissues but cannot develop its specific pathogenic effect in them. *Taphrina cerasi* attacks the buds of cherry trees and stimulates them to abnormal growth. Instead of normal shoots there develop witches' brooms that may reach 2 m. in height and which are characterized by greatly increased branching, lack of flower buds (sterility), and by negative geotropism. In this case, therefore, the buds are both the point of entry and the site at which the pathogenic effects appear.



A similar case is found among the rust fungi, e.g. *Uromyces pisi*, which has its asexual generation on leguminous plants and the sexual generation on various spurge. The hyphae penetrate into the unfolding buds on the rhizome of the spurge, and grow into the developing shoots which become yellowish in colour and markedly elongated; the leaves become broader and shorter and there is an absence of branching and flower formation (Fig. 51).

Wart disease of the potato (Fig. 187) also results from bud infection. The potato tuber is covered by a skin (just as was Siegfried in his horny skin) through which the myxamoebae of the parasite cannot penetrate. The only weak places are the buds (eyes) and the area immediately surrounding them. If these buds are attacked strongly enough they grow out into deformed shoot systems or even into warty growths. The parasite can also gain an entrance into the green aerial parts, stems, leaves, &c., but here the specific reaction of the host is absent, since this occurs only when the specific mode of infection is used.



FIG. 51. Right: a healthy flowering shoot of Cypress spurge (*Euphorbia Cyparissias*). Left: shoots deformed by *Uromyces pisi*. $\times \frac{1}{5}$. Orig.

(d) Needle Leaves as Specific Entry Points for Pathogens

The hyphae of those pathogens which use needle leaves as specific points of entry do not grow up the shoot, as in the case of seedling and bud infections, but down through it. Thus, during the summer, germ tubes of the basidiospores of *Cronartium ribicola* (blister rust of Weymouth pine) and also of *Cronartium asclepiadeum* (bark blister rust of Scots pine) penetrate through the cuticle and stomata into the leaves of the current or the previous year and, during a period of several months, grow through the vascular bundles into the branch and later into the trunk below. They grow through the phloem and, after 2 years, form pycnidia in the cortex of the branch or trunk. After another year the first flask-shaped, shining, golden-yellow aecidia break through the bark (Fig. 52). In this case also, therefore, the place where the disease becomes apparent is far removed organically from the site of infection.

(e) Root-hairs as Entry Points for Pathogens

The root-hairs and the young epidermal cells that give rise to them are not cutinized and are, therefore, accessible to parasites which cannot perforate the cuticle and which would otherwise be dependent on wounds. Thus, the nodule bacteria of Leguminosae (Fig. 179) and the myxamoebae

of club root (*Plasmodiophora brassicae*, Fig. 53) enter their hosts through the root-hairs and the youngest epidermal cells. This mode of infection is also of practical importance for *Fusaria* and bacteria although it is not always easy to prove its occurrence with certainty because of technical

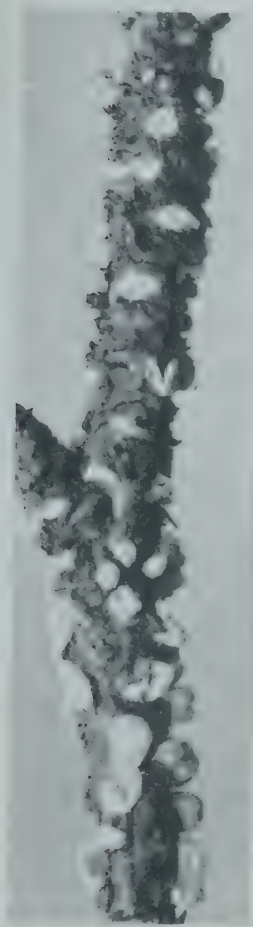


FIG. 52.

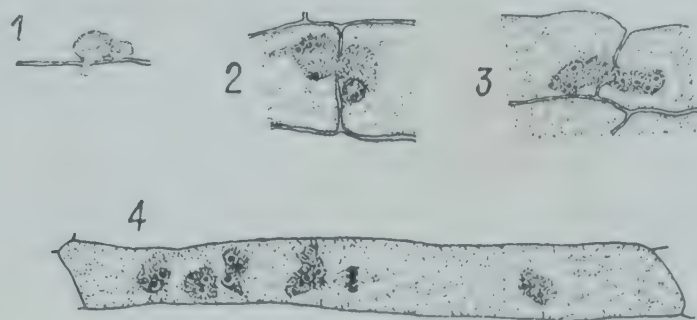


FIG. 53.

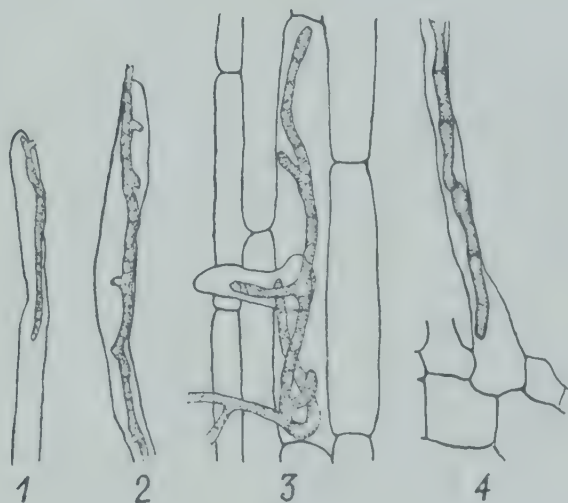


FIG. 54.

FIG. 52. Aecidia of *Cronartium asclepiadeum* (bark blister rust of fir), nat. size. (Orig.)

FIG. 53. Infection of the roots of a cabbage plant by *Plasmodiophora brassicae* (club root). 1 A myxamoeba has made a small opening in the wall of a root hair or an epidermal cell through which it has begun to penetrate into the interior of the cell. 2 and 3 A plasmodium passing through a transverse cell wall. 4 Infected cambial cell with plasmodia; the nucleus of the host cell is dividing. $\times 560$. (After Kunkel, 1918.)

FIG. 54. The penetration of hyphae into root hairs and root epidermis. Explanation in text. 1-3 approx. $\times 100$, 4 $\times 240$. (After Tisdale, 1917, and Watkins, 1938.)

difficulties. Fig. 54, 1, however, illustrates this for *Fusarium conglutinans*, the agent of cabbage wilt in North America; Fig. 54, 2, for *Fusarium lini* (flax wilt); and Fig. 54, 4, for *Phymatotrichum omnivorum* (Texas root rot). The hyphae can also bore directly through the epidermal cells of the youngest parts of the root (Fig. 54, 3) or, in the case of the *Fusaria*, enter the aerial parts through the stomata. In these latter cases, in contrast to the nodule bacteria and the club root pathogen, the root-hairs are not the only possible entry point but merely the more usual one.

4. Entry through Injuries

Infections which take place through injuries are known as wound or traumatic infections, and their causal agents as wound parasites, although this term is not very appropriate. In human medicine, a true wound parasite is, for example, the tetanus bacillus, the cause of lock-jaw, which lives as an anaerobe exclusively in the wound and thence damages the nerve tracts. In this same sense the aerobic plant parasite of crown gall (*Bacterium tumefaciens*) may be regarded to some extent as a wound parasite since it produces a local gall in the area immediately around the wound whilst the more remote tissues of the host are unaffected. In contradistinction, the other phytopathogenic micro-organisms to be considered here use wounds merely as entry points and passage ways, and their pathogenic development occurs mainly in a totally different part of the plant. They are, therefore, not wound parasites in the strict sense of the word, just as those micro-organisms which enter through the natural openings are not called stomatal parasites: but the term persists and it is not desirable to coin a new one.

Many micro-organisms included here are facultative wound parasites. They are specialized in their mode of attack, for example, through the skin or stomata, but breaks in the skin, i.e. wounds, can also be used equally well. Thus, *Botrytis cinerea*, the classical example of mechanical perforation of the cuticle (Fig. 1), can also enter through wounds.

Again, other micro-organisms are obligate wound parasites. For example, the stem parasites of our forest trees are unable to penetrate the thickened outer layers, cuticle, periderm, &c., and are therefore dependent on wounds for ingress.

Conversely, there are some pathogens that cannot infect through wounds. In certain mixtures of viruses of the potato, e.g. crinkle and paracrinkle (Fig. 155), the components may be separated by needle inoculation because the one moiety, e.g. the X-virus, enters the experimental plant through the wound whereas the other moiety, the Z-virus, is dependent on grafting as a specific mode of infection and, therefore, remains behind (Salaman, 1930).

It is not necessary for the wound to be visible to the naked eye since microscopically small holes, cracks, cuts, &c., are sufficient. Many viruses, e.g. the X-viruses of potatoes and the virus of tobacco mosaic, can be introduced into healthy plants by rubbing the latter with cell sap containing the virus. During this manipulation hairs and epidermal cells are injured and through these abrasions wound infection takes place.

Phytophthora infestans (late blight of potato, Fig. 195), *Alternaria solani* (early blight of potato, Fig. 216), and *Plasmopara viticola* (downy mildew of vine), although normally making use of the stomata on the underside of the leaves as entry points, can also penetrate into the interior of the leaf from the upper side by means of cracks in the cuticle. The first two can also use small cracks in the periderm of the tubers as points of ingress. Similarly, *Bacillus atrosepticus* (syn. *Bac. phytophthorus*), the cause of

black leg of potato (Fig. 298) and a wet rot of the tubers, usually enters through an injury in the region of a lenticel and only rarely through the eyes. This is because the mutual pressure of tubers in storage very easily damages cell walls in the region of lenticels.

The extent of injury is of little consequence in itself, but indirectly it is significant because small wounds heal more quickly than large ones and are thus available to the pathogen for a shorter time. For the same reason the broken hairs mentioned above lose their capacity for allowing the entrance of the virus extract after a few minutes. In Fig. 55 the leaves of *Nicotiana glutinosa* were rubbed with a finger and one half of each leaf was sprayed immediately with aucuba mosaic virus whilst the other half was sprayed after 1, 2, and so on up to 30 minutes (abscissae). The ordinates give the number of lesions in the delayed spraying compared with the number of successful infections on the half-leaf that was sprayed immediately. Even after 2 minutes only 72% of the inoculations are successful, after 5 minutes 44%, and after 30 minutes only 2.8%.

Such traumatic entry points are caused mainly by various mechanical and physical injuries due to hailstorms, feeding animals, and various human activities.

Coniothyrium diplodiella (white rot of grapes) is known in western Switzerland as the 'hail fungus' but obviously every hailstorm that beats down on the maturing grapes is not followed by an epidemic of white rot. However, the fact that the vineyardist, on the basis of his local observations, combines the two phenomena in the name of the disease emphasizes the damaging effect which he attributes to the hail as a precursor of *Coniothyrium*. In the same way, hailstorms facilitate attack on ripe cherries by *Monilia cinerea*, which causes the dreaded brown rot, and attack on silver fir by *Cylindrocarpon cylindroides* (twig canker). In general all wound parasites can to some extent use injuries caused by hail as points of attack.

Damage to the roots of spruce and fir by rodents and also, perhaps, wounds on the trunks caused by red deer provide points of attack for *Trametes radiciperda* (red rot or butt rot). The feeding-places of the olive-brown, pine bark moth (*Grapholita pactolana*), as well as injuries due to hailstorms and frost, facilitate attack by *Nectria cucurbitula*, which causes spruce die back.

As an example of wounds caused by man injuries due to scraping may be instanced; these occur in forests in which the trees are of different ages and the felled trees scrape or strip the younger standing trunks (felling damage). These injuries serve as entry points for both *Trametes radiciperda* (red rot) and *T. pini* (ring rot) of spruces and pines (Fig. 295), and

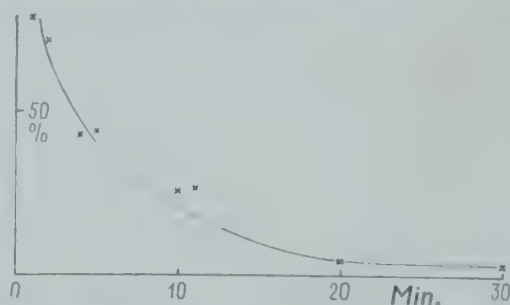


FIG. 55. The period during which the smallest wounds allow entrance to aucuba mosaic virus. Explanation in text. (After Sheffield, 1936.)

for many other tree-destroying fungi. Further, the wood-damaging effect of collecting debris is due not only to the removal of humus-forming material but also to the mechanical injury caused by iron rakes to shallow lying pine roots which opens the way to their attack by *Trametes radiciperda*.

Cuts and artificial wounds which facilitate attack in both *Trametes* spp. are caused in 'green pruning' of living spruce and pine branches when there is poor natural branch cleaning. Breaking of such branches by snow has the same effect. Green pruning of oaks facilitates attack by *Daedalea quercina*. In the grafting and pruning of fruit trees cut wounds are made which allow of the entrance of *Bacterium tumefaciens* (crown gall, Fig. 288).

Finally, man himself causes the injuries necessary for the attack of many parasites during the harvesting and transporting of fruit, sugar beet, and potatoes; for example, the bacteria causing wet rot of potatoes enter through injuries produced at lifting.

5. Entry through Lesions caused by Parasites

Through its local disease focus a primary parasite can give rise to a damaged area which can be used as a point of entry by a secondary or following parasite, and may be used for an initial saprophytic phase. This enables the secondary parasite to invade the host organism and to spread out from the primary focus to attack the healthy, living host tissue. In other cases the primary pathogen does not provide the secondary one with an entry point but weakens the host in certain ways; for example, it decreases the resistance of the host and makes it possible for the secondary weaker parasite to infect it. The latter case will be discussed in Chapter 4 in connexion with the disposition of the host; here we shall confine our attention to the former condition, i.e. the production of entry points by parasites.

Fusarium caeruleum (dry rot of potato) cannot penetrate the uninjured, superficial periderm of the tuber; even small cracks in the skin can be used only with difficulty because the direct transition from spore germination to a parasitic way of life does not take place readily. But, if the tubers are already attacked by blight (Fig. 271) caused by *Phytophthora infestans*, which gains an entrance through the lenticels and small cracks in the skin of the tuber, then *Fusarium caeruleum* finds in the necrotic tissue not only an entry point but also the necessary substrate from which the healthy tissues of the tuber can gradually be invaded, and the tuber finally mummified. The incidence of dry rot is often directly related to the incidence of blight attack.

Nectria galligena (apple canker, Fig. 287) may enter directly through wounds in the bark but it more usually invades through old scab lesions caused by *Endostigme inaequalis*. Thus, by controlling the scab the canker can also be indirectly suppressed.

Witches' brooms of silver fir (*Melampsorella caryophyllacearum*, Fig. 230) are in themselves harmless and are regarded as natural curiosities. But when infection takes place in the main shoot the mycelium grows into

its bark and cambium and, during the course of a year, produces a cracked stem canker. These cankers serve as entry points for secondary wood-destroying fungi, especially *Polyporus Hartigii* (white rot) and *Agaricus adiposus*, which produces a characteristic shell-like brittleness. In forestry the great importance of witches' brooms of silver firs lies in the trunk or timber diseases which result from these secondary parasites.

6. *Entry through Wounds caused by Vectors*

Many disease germs which cannot attack uninjured plants are conveyed from diseased to healthy individuals on the piercing or sucking mouth parts of insects (wound transmission). This is a special form of wound infection in which the insect making the wound is, at the same time, the vector which transmits the parasite to the new host. This group will be discussed in the following chapter ('Epidemiology') in connexion with the spread of disease agents by insects.

§ 6. The Colonization of the Host

The colonization of the host by the parasite introduces the second stage of infectious disease (p. 5), that of incubation. During this phase the pathogen spreads to a greater or lesser extent from the site of infection into the host organism, i.e. it proceeds from local infection to general infection. Only if colonization is successful does infection constitute the first step in pathogenesis; only then can it lead to disease, otherwise it proves abortive.

In plant pathology the process of colonization raises a number of questions among which are the following:

1. How does the pathogen proceed to colonize the host? (1. 'The Method of Colonization of the Host'; p. 57).
2. By what route does the pathogen spread in the host? (2. 'The Paths of Spread of the Pathogen'; p. 61).
3. How long does it take the pathogen to spread in the host? (3. 'The Rate of Spread of the Pathogen in the Host'; p. 66).
4. How far does the pathogen spread in the host? (4. 'Local Infection and General Infection'; p. 68).
5. Where does the pathogen go to in the host? (5. 'The Selective Colonization of the Host'; p. 70).

1. *The Method of Colonization of the Host*

Most of the parasites which cause infectious diseases of plants pass through a saprophytic stage to a parasitic one during their attack on the host. Thus, at first the germ tube of a spore germinating in an infection drop (Fig. 1) lives half saprophytically because it obtains only a part of its nutriment from the mother cell, the rest comes from the infection drop. It is the infection hypha which effects the transition to parasitic feeding, i.e. to feeding on the substance of the host.

Most parasites carry out the conversion easily, their vitality being

sufficiently great to enable them to attack the uninjured host directly without first damaging it by toxins, &c. (direct assault on the host). Thus they feed on the living substance of the host, i.e. they are biotrophs.

The zoospores of *Phytophthora infestans* (late blight of potato) at first swim about for a time in the infection drop but then come to rest and form a membrane about themselves. They then produce a germ tube which penetrates the outer walls of the epidermal cells, in the way shown in Fig. 1 (i.e. if they do not enter by a stoma), and begin to absorb the content of the invaded cell whose protoplasm soon coagulates. The infection hypha then pierces the inner cell wall and spreads farther in the middle lamella between the cells (Fig. 56, 1), from time to time producing fine haustoria which enter the adjacent host cells. After a short time the contents of the latter become brown and the cells die.

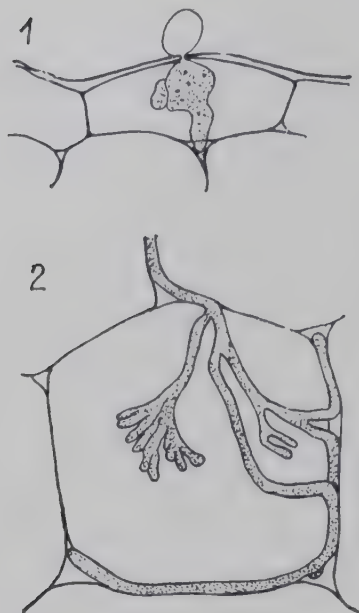


FIG. 56. The spread of *Phytophthora infestans* (potato blight), in tomato tissues. 1 A zoospore has germinated on a leaf and after 5 hr. has already penetrated through the epidermal cell into the intercellular system. 2 Intracellular branching and intercellular spread of an infection hypha in the flesh of a tomato fruit. 1 $\times 500$, 2 $\times 340$. (After Röder, 1935.)

The attack by this fungus is not preceded by a long saprophytic stage and there is no preparatory long-range effect; the attack follows directly on the germination of the spore.

A second group of disease germs, including many pathogenic fungi, are unable to effect this direct transition from spore germination to the parasitic way of life at all easily; they must first obtain a saprophytic foothold on the protoplasmic cell contents liberated during the breakdown of the tissue or on tissues killed as the result of a bruise or wound. During this introductory saprophytic phase they gain strength and acquire the necessary vitality to overcome the resistance of the host and to live on or in it as true parasites.

These pathogens, like those of the first group, also attack the host cells directly without producing an obviously damaged zone in advance of themselves, i.e. they also are biotrophs. At the margin of the infection focus intact cells are thus contiguous with cells which have been damaged or killed by the fungus (Fig. 57). The parasitic phase cannot ensue directly from germination and the organisms must first gain strength by living as saprophytes, i.e. there is direct attack following an introductory saprophytic stage.

Many wound parasites belong to this group (p. 54). In artificial infection experiments the substrate for the necessary saprophytic stage is provided by killing small areas in inessential tissues either by burning them with glowing copper wire or by freezing them with liquid air, &c. After these preliminary treatments infection takes place readily whereas, in their absence, it would succeed only occasionally; this is true of the two cases

already mentioned, namely, *Hysterographium* on ash and *Coniothyrium diplodiella* (p. 55) on branches of grape vine.

Finally, there is a third group of pathogens which rarely or never feed directly on living plant substance because the toxic secretions of their hyphae

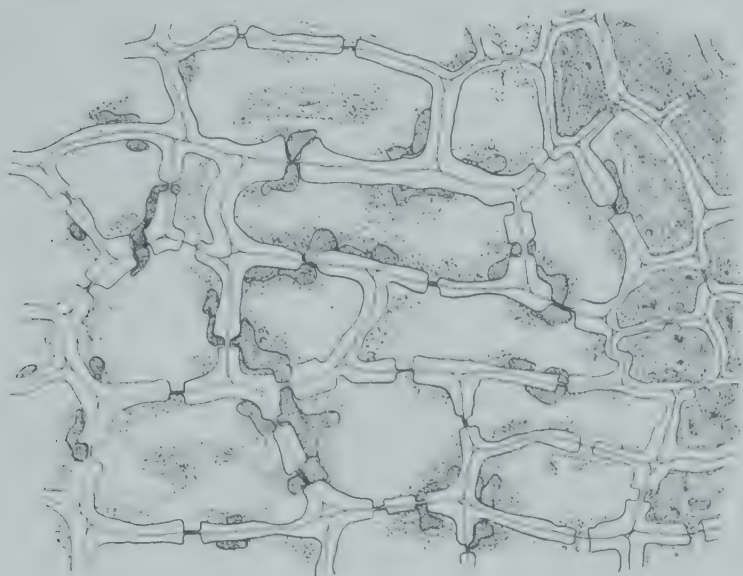


FIG. 57. Section through a 4-5-year-old ash branch attacked by *Hysterographium fraxini*. Left: dead cells of primary cortex colonized by fungal hyphae. Right: healthy cells. $\times 750$. (After Zogg, 1943.)

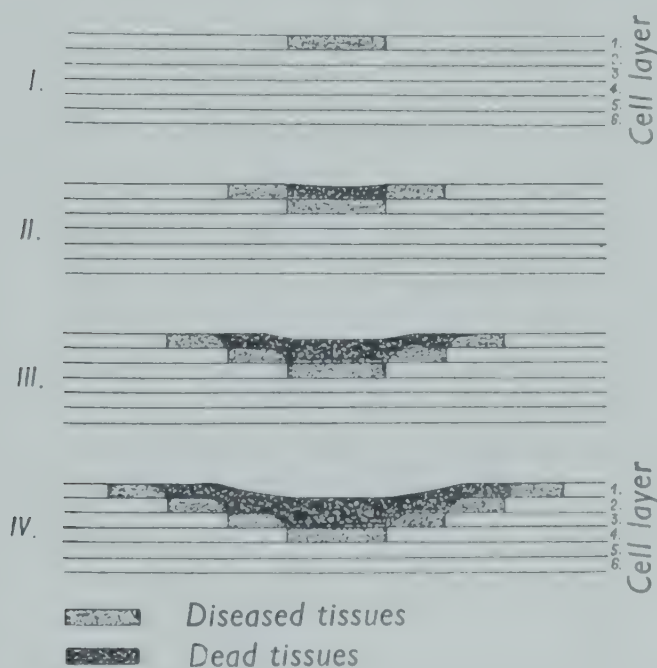


FIG. 58. The mode of infection of the roots of a potato plant by *Corticium vagum*. Explanation in text. (After K. O. Müller, 1924.)

kill the host cells in advance of them, i.e. the parasite attacks indirectly. These pathogens feed on the dead substance of the host, i.e. they are necrotrophs.

Thus, *Corticium vagum* (syn. *Rhizoctonia solani*), which causes black

scurf of potato, forms, like the agent of eyespot of wheat (see Fig. 59), hyphal strands on the root surface which press themselves against the epidermis like appressoria. The underlying epidermal cells are poisoned by the metabolic products which diffuse out of the fungus, sicken, and die (Fig.

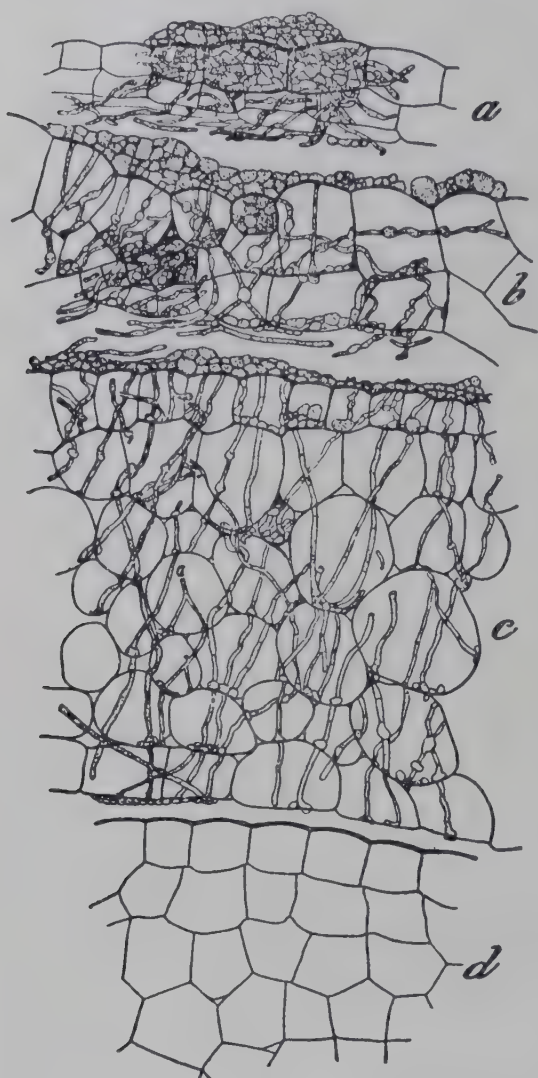


FIG. 59. The colonization of a wheat shoot by *Cercospora herpotrichoides* (eye spot). Transverse section cut 7 weeks after infection. The fungus has already permeated the leaf sheaths *a*, *b*, and *c*; it first forms a stromatic plate on each and then penetrates into the deeper tissues. $\times 500$. (After Sprague and Fellows, 1934.)

58, stage *I*); the fungus then enters them. This process is repeated in the deeper cell layers of the cortical parenchyma, &c. (Fig. 58, stages *II-IV*), so that the fungus grows successively through the vascular bundle into the pith parenchyma.

During their spread in the host tissues these parasites are always surrounded by a zone of dead cells, from beginning to end, therefore, they feed saprophytically and reproduce themselves exclusively in dead tissues. Their influence can sometimes also be detected microscopically by the disintegration of the cells or their maceration, because not only their toxins but also their cell wall dissolving enzymes diffuse in advance of them and break down the tissues.

These perthophytes (i.e. parasites which first kill the tissues and then colonize them; Münch, 1929) act, therefore, like toxigenic or pathogenic saprophytes. They are really saprophytes, i.e. they are necrobionts. Strictly speaking, the infection locus is an island of dead tissue in the middle of the living host organism; on this island the pathogen feeds saprophytically and from it carries its attack still farther into the remaining intact host tissues.

This group includes a large number of plant pathogenic fungi, wound parasites as well as species which attack directly. Examples are *Leucostoma Persooni* (syn. *Valsa leucostoma*), *L. cincta*, and *L. nivea*, the cause of die back (*Apoplexie*) of apricot, cherry, &c., and various *Nectria* spp., which cause the coral spot disease of many deciduous and evergreen trees. As a rule, in this group the 'taking' of infection is facilitated by antecedent killing of small areas of tissue.

This third group is of scarcely any importance among the parasites

which cause disease in man. *Bacillus botulinus* (food poisoning) is a true toxic saprophyte; it lives saprophytically on dead flesh and its poisonous metabolic products enter into our digestive system. *Bacillus tetani* (lock-jaw) is a perthophyte; it first colonizes the dead tissues of the wound, from them damages the host organism, and then continues to invade so long as its anaerobic capacity permits.

These three behaviouristic groups are, of course, sharply distinguishable only in ideal cases such as the typical examples given. In reality, most

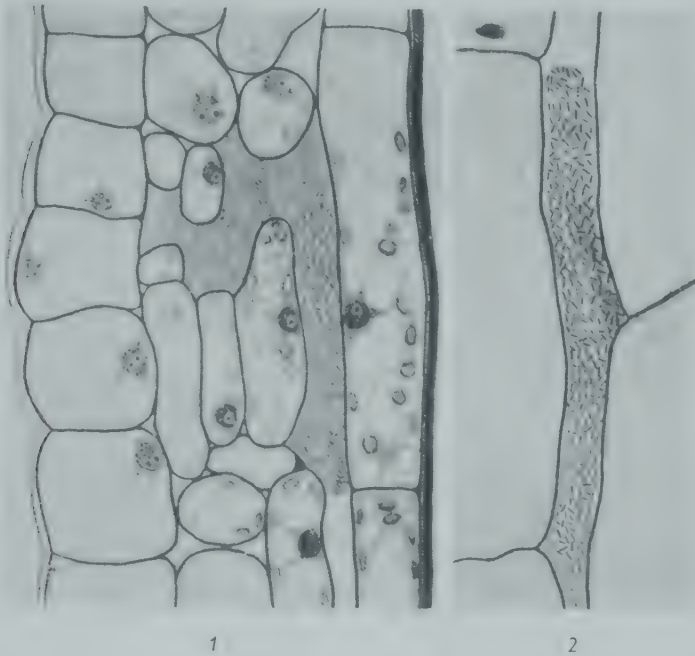


FIG. 60. The path of spread of *Bacterium tumefaciens* (crown gall) in the tomato stem. 1 Zoogloea which is forcing itself into the hypodermis through the intercellular spaces. Extreme right: thick-walled collenchyma begins. 2 Zoogloea in the intercellular spaces of the pith. $\times 830$. (After Hill, 1928.)

pathogens can, to a greater or lesser extent, adopt one or other procedure according to their vitality, the environmental conditions, and the reactivity of the host. It will be seen later (Chapter 4) that one and the same strain of cereal rust may exhibit a complete series ranging from absence of toxic effect (first group) on a susceptible cereal variety, to extreme toxic effects on a hypersensitive variety (third group).

2. The Paths of Spread of the Pathogen

Primitive parasites, especially many perthophytes, do not follow any special route of spread; they grow through the host tissues without regard to their histological differentiation and, consequently, they spread irregularly. An impression of this type of advance is given in Fig. 18 in which *Pseudomonas campestris* is dissolving out a large cavity in the tissues of a cabbage leaf during the first stages of attack.

With refinement of its parasitic faculties a pathogen discriminates in its behaviour towards (a) host cells, and (b) host tissues.

(a) *Differential Behaviour of the Pathogen towards Host Cells*

Some parasites, such as viruses and the agents of wart disease of potato (Fig. 187) and club root disease (Fig. 128), exist and spread entirely within the cells of the host, i.e. they are exclusively intracellular. The

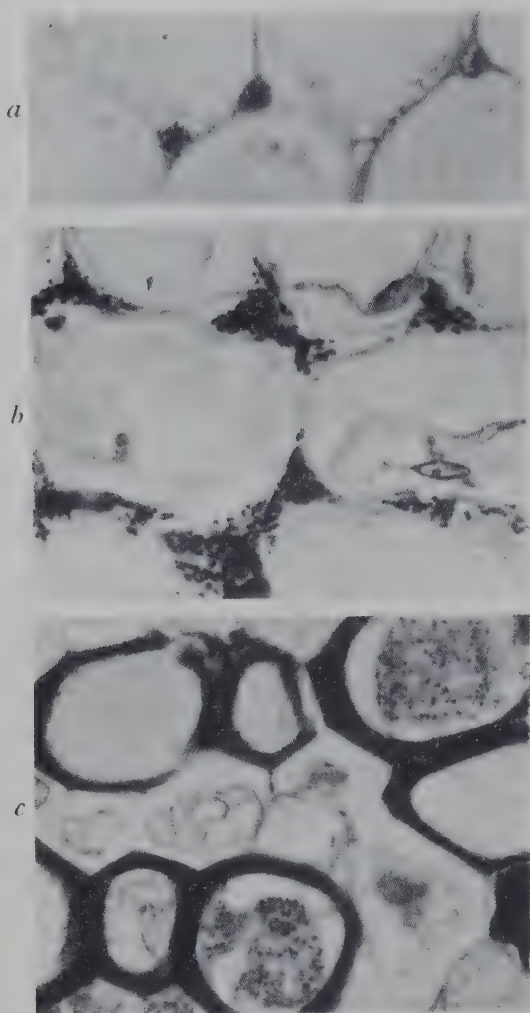


FIG. 61. Stages of infection by *Bacterium malvacearum* (angular leaf spot disease of cotton). *a* initial stage, bacteria only in the intercellular spaces. *b* later stage, bacteria also in the middle lamellae between the cells. *c* late stage of a severe infection, the bacteria have penetrated into the stem tracheids. Approx. $\times 500$. (After Stoughton from Gäumann, 1932.)

plasmodia of the fungus causing wart disease of potato never leave cells which they have once invaded; in contrast, the plasmodia of the club root fungus migrate directly from cell to cell by perforating the cell walls. It seems likely that viruses spread via the plasmodesmata because stomatal guard cells, which are not connected to the adjoining cells by plasmodesmata, do not become infected by tobacco mosaic virus (Sheffield, 1936). Possibly, virus particles can also penetrate through the sub-microscopic pores of the cell walls which measure approximately up to $10\ \mu\mu$. Finally, parasitic fungi generally tend to make their way through the pits in the cell walls, especially in woody tissues (Fig. 57).

Certain other parasites grow only between the host cells, i.e. intercellularly, dissolving the pectin of the middle lamellae (cf. Figs. 9 and 60). Still others spread intercellularly during the early stages of infection but invade the host cells themselves in later stages of the disease (cf. Fig. 61); this order is reversed in the case of the root nodule bacteria of the Leguminosae (Fig. 179) which are, at first, intracellular but later intercellular.

Still other parasites, such as *Pythium de Baryanum*, one of the pathogens causing damping-off (*Wurzelbrand*) of

seedlings, behave indifferently in this respect. According to the vitality of the host and the kind and age of its tissues they will grow freely either within or between the host cells.

(b) *Differential Behaviour of the Pathogen towards Host Tissues*

Some pathogens, amongst them *Pythium de Baryanum* and *Phytophthora infestans*, are non-selective and permeate all tissue systems, epidermal, ground, vascular, storage tissue, &c. Other parasites, however, are selective

in their path of spread, in particular the vascular bundles are used as avenues or channels of infection. This is analogous to the use made by human pathogenic micro-organisms of the circulatory system as a means of transport and a route of spread. Such micro-organisms may favour either the blood or the lymphatic system; thus, streptococci and anthrax bacteria are distributed mainly by the blood stream, whilst tetanus toxin and the polio virus are transported in the lymphatic vessels. An analogous distinction is found among plant pathogens which may be restricted specifically to either phloem or xylem as a route of spread.



FIG. 62.

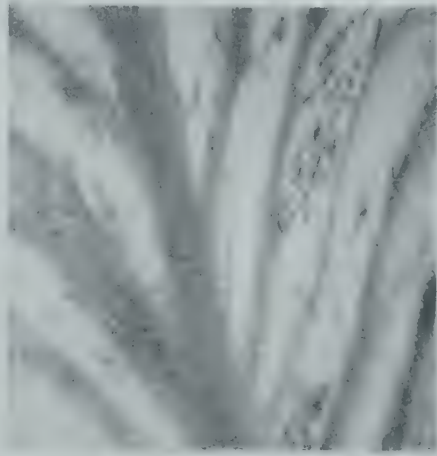


FIG. 63.

FIG. 62. Early stage of the ring rot of potato tuber (*Bacterium sepedonicum*). By hand pressure some of the rotting matter has been squeezed from the vascular zone. (After Stapp, 1933.)

FIG. 63. Black rot of cabbage (*Pseudomonas campestris*) from a diseased cabbage leaf, photographed by transmitted light. The bacteria first permeate the ground tissue (as shown in Fig. 18). As soon as they reach the vascular bundles they use these almost exclusively for further spread. Approx. nat. size. (After Stapp, 1939.)

Amongst the fungi which spread via the xylem (tracheomycoses, hadromycoses) are *Fusarium lycopersici* (wilt disease of tomato) and *Verticillium albo-atrum*, the causal agent of a wilt disease of potato (Figs. 244 and 285) and other herbaceous plants (Rudolph, 1931). Among bacteria (tracheobacterioses) the following may serve as examples: *Bacterium sepedonicum* (ring rot of potato, Fig. 62), *Pseudomonas campestris* (black rot of cabbage, Fig. 63), and *Bacterium solanacearum* the causal agent of wilt in tobacco and numerous other tropical cultivated plants and weeds.

Some of these micro-organisms cause a brown discoloration and a partial dissolution of the vessels (Fig. 64) along their tracks or routes which, for this reason, often become visible to the naked eye.

The phloem or sieve tubes serve as the path for most viruses (Fig. 65), e.g. leaf roll and X-viruses of potato, a specialization which appears to be necessary because of their close relation with the protein compounds of the host.

However, in the later stages of infection all these pathogens, fungi,

bacteria, viruses, break out from their specific distribution channels and extend into the ground tissues of the host where, usually, they attain their maximum pathogenic activity. Hence, they are not confined to the vascular bundles and do not cause diseases specific to the vascular tissues, i.e. they are not 'vascular diseases' in the strict sense, which might roughly be compared with malaria which is a specific malady of the blood. The position is

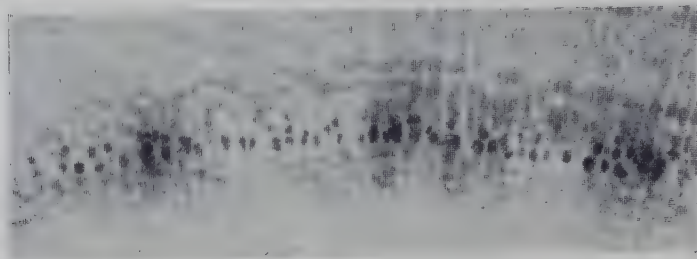


FIG. 64. Typical discoloration of the large vessels of the spring wood of *Ulmus americana* (American elm), after serving as paths of spread for *Ophiostoma ulmi* (Dutch elm disease). $\times 5$. (After Banfield, 1941.)



FIG. 65. Progressive colonization of tobacco leaves by the Us-strain of potato X-virus beginning from the petiole. Approx. $\times \frac{1}{3}$. (After Köhler, 1942.)

rather that the conducting strands are favoured simply as the channel of most rapid and efficient spread within the host.

The usual direction of advance along these specific routes of colonization is predetermined by the direction of flow of sap. Fungi and bacteria migrate more quickly in an acropetal direction, i.e. towards the apex, because in the vessels they are favoured or carried by the rising sap; the movement of viruses, on the contrary, is quicker in a basipetal direction, since they descend with the flow of assimilates in the phloem (Fig. 66).

The method whereby the germs of infection travel along their routes of extension may be either the active progressive growth of the pathogen itself or its passive transport by the host.

In the first group, which includes the mycelial fungi, the parasite grows

actively through the host by its own power, using a penetration mechanism similar to that of infection (Fig. 1). In plant disease, in contrast to human pathology, this is by far the larger group.

In the second group the host supplies the 'motive power' by means either of its vascular flow, its mechanism of cell division, or its proliferation of specific tissues.

Some bacteria and a few fungi are transported purely mechanically in the sap flowing through the xylem. Under optimal conditions, and using *Datura Tatula*, which as an experimental plant is peculiarly suitable for this purpose, it has been found that even *Bacterium tumefaciens* can be carried for a distance of 120 cm. in the course of a few weeks. This is the reason for the frequent failure to eliminate infections of the crown gall disease of fruit trees by pruning away the affected portions of the root. Occasionally it may happen that up to 35% of the young trees prove to be still diseased and form new tumours (Stapp and Müller, 1938). Conidia of *Ophiostoma ulmi*, the agent of Dutch elm disease, may be carried upwards by the sap stream for 5 m. in 24 hours in the large vessels of the spring wood (Banfield, 1941).

Cell divisions of the invaded host cells serve, for example, to pass the plasmodia of club root disease (Fig. 53) and the root nodule bacteria of certain Leguminosae directly and passively from mother to daughter cells.

Passive transport of the pathogen by specific tissue proliferations of the host (cell strands) is found, for example, in crown gall of plants. Thus, if the primary tumour originates in young unligified tissue of the host, portions of tissue in the xylem region may start to proliferate, forcing themselves forward between the host tissues, especially the vascular bundles, by sliding growth. They may then form secondary tumours some distance away. Fig. 67 depicts a stem of *Chrysanthemum indicum* which was inoculated with *Bacterium tumefaciens* at *x-x* by puncturing with a needle, and was thus induced to form the primary tumour *t*. A tumour strand (*Krebssträhne*) grew from this up into the leaf region; on the edge of the leaf, immediately adjoining, it formed only a small secondary tumour *A*. But in the case of the leaf 2 cm. higher, the strand grew right through the midrib, being recognizable externally by the swellings

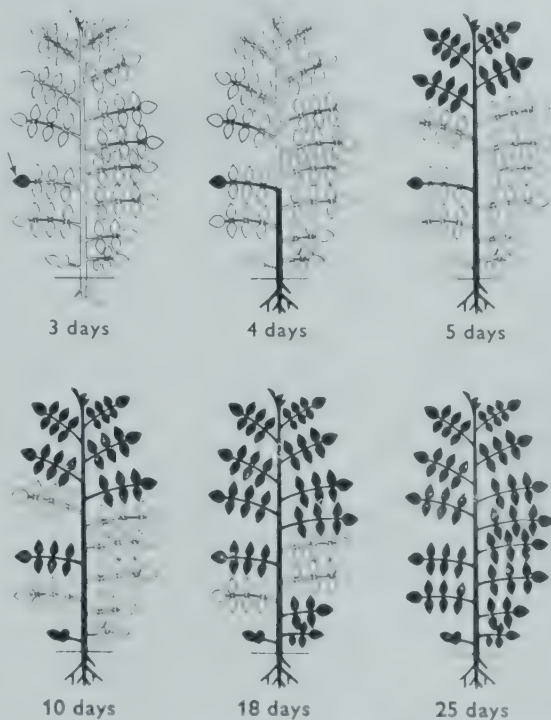


FIG. 66. Progress of spread of tobacco mosaic virus (black) in a tomato plant, approx. 45 cm. in height. The inoculated leaflet is marked by an arrow. (After Samuel, 1934.)

produced; it forked and entered both halves of the divided leaf. This strand gave rise in all to six smaller secondary tumours (*B*, *X*, *Y*, &c.), of which the farthest was 9.5 cm. removed from the stem. The apex of one such advancing strand is seen in Fig. 68, 1, whilst Fig. 68, 2, shows part of a strand in a different experimental plant.

This method of internal infection by means of cellular strands carrying bacteria has been the subject of much discussion, largely because it some-



FIG. 67. Primary and secondary tumours on *Chrysanthemum indicum* caused by *Bacterium tumefaciens*. Explanation in text. Approx. $\times \frac{1}{4}$. (After E. F. Smith *et al.*, 1912.)

what resembles the formation of metastases in human carcinoma. We shall return to this in connexion with the problem of galls in Chapter 5.

Finally, the way in which viruses migrate is still uncertain. It seems likely that they are transported, at least partly, through the phloem in the stream of anabolites. It is possible that some do not migrate at all, but grow through phloem and plasmodesmata in the form of long polymerization chains. The spread of viruses is also curiously discontinuous (Köhler, 1942), so that often strips of the stem remain free from the virus (Stapp and Marcus, 1943).

3. *The Rate of Spread of the Pathogen in the Host*

The rates of spread of pathogens inside the host differ in the two groups considered above, i.e. those invading by active growth and those transported passively by the host.

The rate of spread of actively growing fungal pathogens, i.e. most of the parasitic mycelial fungi, is directly related to the hyphal growth-rate. Fungi which grow quickly are also capable of making rapid progress within the host. Thus, rhizomorphs of the honey agaric, *Armillaria mellea*, may grow on artificial media, and probably also on affected boles, as much as 18 mm. per day, and *Stereum purpureum*, which causes a white rot, may grow inside the poplar as much as 13 mm. daily. In contrast to these, in



FIG. 68. Tumour strand in the vascular bundles of *Chrysanthemum indicum*.
1 approx. $\times 120$, 2 approx. $\times 200$. (After E. F. Smith *et al.*, 1912.)

Trametes pini (Fig. 295) the rate of advance inside pine trunks averages only 18 cm. per annum (Bavendamm, 1928; Münch, 1932).

The rate of spread of such fungi within the host, like their growth-rate in artificial media, is closely correlated with temperature (cf. Fig. 69). We shall return to the question of the influence of temperature on the parasitic adaptation of pathogens in Chapter 3.

In the case of passively transported organisms we must distinguish between long-distance transport in specific channels and intensive colonization of tissues, e.g. ground tissue.

Depending upon the rate of protoplasmic streaming, long-distance transport in specific channels occasionally takes place with astonishing rapidity, and generally much exceeds the active extension of parasitic fungi by their own growth. Some average distances passed through in the course of one hour are as follows: curly top virus of sugar beet, 152 cm. (Bennett, 1934); stripe mosaic of maize, 20 cm. (Storey, 1928); and tomato mosaic, 18 cm.

(Kunkel, 1939). The rates at which pathogenic bacteria, viruses, &c., spread in the fluids of the human body are of the same order; thus, spirochaetes and trypanosomes can be demonstrated in regional glands a few minutes after inoculation; anthrax bacteria will reach the lungs, liver, and spleen of experimental animals one hour after inoculation through an epidermal lesion.

Short-distance infection from cell to cell is, however, very slow in the plant pathogens referred to above, being $10\ \mu$ per hour for one and $40\text{--}80\ \mu$ for another. Valid comparisons with animals cannot be made here since their cells are not enclosed in rigid walls of cellulose, &c.

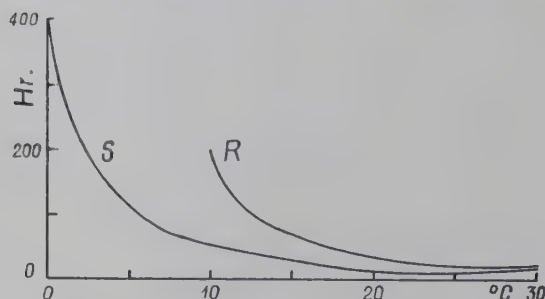


FIG. 69. Rate of spread of *Sclerotinia fructigena* (curve *S*) and of *Rhizopus nigricans* (curve *R*) in the flesh of peaches at different storage temperatures. Ordinates: time in hours until the rot has exceeded the marketable limit. If kept at 3°C . in a refrigerator truck, transport may be extended up to 10 days; at a summer temperature of 25°C ., however, the fruit must be marketed by the next day. (After Brooks and Cooley, 1928.)

4. Local Infection and General Infection

How far does the pathogen spread in the host? Two extremes are possible: local infection and general infection.

In local infections the parasite colonizes only the point of entry and is confined to this and to the immediately adjoining tissues. According to the duration of the infection and the range of its patho-

genic influence, local infections fall into two groups: local infections in the strict sense and infection foci.

Local infection, in the strict sense, represents the single occurrence of a usually acute disease, whose pathogenic effect does not extend much beyond the area occupied by the parasite, i.e. there is a short-range effect. Examples are cereal rusts (Fig. 166), blight of potato (Fig. 195) and downy mildew of vine, leaf spot diseases (Figs. 215, 216), smut of maize (Fig. 144). and club root disease (Fig. 128).

Local infections comprise the most frequent type of disease in the plant kingdom; this contrasts with what occurs in human pathology, where general infection is the normal type of distribution. True local infections are rare in human pathology but they occur in certain skin diseases, e.g. ringworm.

The causal agents of plant disease do not, as a rule, penetrate much beyond their point of ingress. In certain diseases, such as maize smut and club root disease, the pathogens are strictly confined to the original site of infection for the entire period of their existence. In other infectious diseases, e.g. the first three in the examples given above, dispersion may take place, i.e. new centres of infection may be started. This dispersion, however, is effected from the outside, i.e. externally. The parasite forms spores at the locus of the primary infection (Fig. 100), and these are transported by the

agency of wind and rain to neighbouring leaves, branches, &c., where they originate new centres of infection. Thus, even in cases of local infections, the host may in this manner become thoroughly infected and diseased; for example, a potato plant suffering from late blight (Fig. 195). It should be noted that this effect is not caused by a general infection, but by an accumulation of air-borne local infections.

Through this spatial restriction or localization of the parasite-host relationship, a general infection of the plant body is certainly rendered

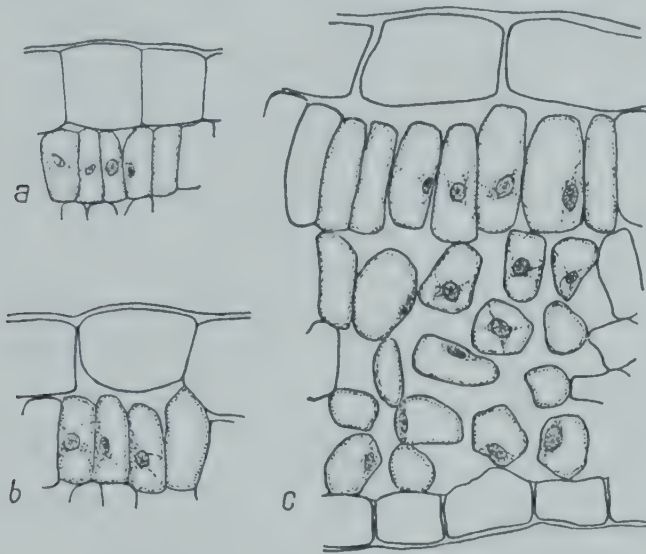


FIG. 70. Separation of the epidermis in leaves of *Victoria plum* with silver leaf disease. *a* Early stage showing cytological defects in the palisade cells. *b* The epidermis beginning to separate. *c* Late stage in a leaf already chlorotic. Chloroplasts omitted throughout. Approx. $\times 300$. (After Tetley, 1932.)

more difficult but, on the other hand, the defence and humoral immunization of the whole body are also rendered more difficult.

Infection foci are associated with chronic disease. Thus, a persistent and often quite inconspicuous local centre will constantly pour out toxic substances and occasionally release batches of the pathogen itself into distant parts of the host organism, in the manner of long-distance infections, and there evoke pathological disturbances.

A classic example is provided by the silver leaf disease of plums and other deciduous trees (species of *Acer*, *Carpinus*, *Sorbus*, &c.) caused by *Stereum purpureum*. When local infection occurs in the roots or trunk, toxins produced by the pathogen and necrogenous substances from the injured tissues travel away from this centre for many metres in the flow of sap. They are carried to distant portions of the trunk and crown and produce the actual disease, at the earliest, one year after infection. This long-distance effect, similar in type to that of tetanus, leads to a brown discoloration of the woody parts, and a pathological change in the leaf structure which results in a silvery effect. These symptoms are caused by two different chemical principles: the browning of the tissues by one which is thermostable, and

the silvering effect by one which is thermolabile (Brooks and Brenchley, 1931).

The thermolabile principle causes the epidermal cells of the leaves to swell, the effect beginning near the veins and spreading into the interveinal areas. The cells then separate from the palisade tissue, and the resultant air-filled cavities give rise to the characteristic silver sheen. Hypertrophy of the spongy mesophyll cells and enlargement of their intercellular spaces enhance this effect. Simultaneous pathological changes take place in the nuclei, cytoplasm, and chloroplasts, and in severe cases lead to the death of the affected tree within a few years.

Thus, the focus of the disease lies at the base of the stem, whilst the injury is in the peripheral organs, the foliage; the vessels form the connecting link, analogous to the human blood stream.

Among others, the wilt diseases mentioned on page 63 also belong to this group. These pathogens colonize primarily the vascular bundles through which they gradually spread upwards. Toxic substances are carried far ahead of them into the peripheral regions, again mainly the foliage, where they produce the characteristic pathological symptoms long before the pathogen itself has reached the tissues concerned. In fact, these tissues are colonized by the parasite only after their physiological breakdown has occurred.

In contrast to the pathogens causing local infections, which are permanently confined to the more or less immediate neighbourhood of their point of entry and which spread from this area only externally, the pathogens which cause general infections continue their colonization immediately after their successful invasion of the host. Initial infection is followed at once by extensive generalization, the primary focus spreading through the interior of the host organism.

In plant pathology such general infections are usually called systemic; examples are *Uromyces pisi* (Fig. 51), several *Verticillium* diseases (Figs. 244 and 285), bacterial diseases (Fig. 61), and virus diseases (Fig. 155). These pathogens eventually colonize the entire plant body with the exception of the growing-points. General infection is followed by generalized disease; specialization is found at most in the initial route of extension of the parasite. But in one particular group of general infections the manifestation of the infection is restricted to certain tissues or organs, i.e. there is local disease; we shall discuss this group immediately.

5. *The Selective Colonization of the Host*

Where does the parasite settle down in the host or, alternatively, to what part of the host does it spread? The spatial extension or distribution of the disease agent in the host, i.e. the micro-organism in the macro-organism, is as much a problem of the specific susceptibility of the host tissues and organs as it is of the selectivity or 'predilection' of the pathogen for certain tissues. The two aspects cannot be separated from one another.

Some parasites such as *Botrytis cinerea* (Fig. 1) and *Corticium vagum*

(Fig. 58), colonize and destroy all kinds of tissues and organs and all kinds of hosts without distinction or selection, providing that the external conditions allow them to attack. The apple scab fungus (*Endostigme inaequalis*) is able to attack quite different host organs, twigs, leaves, and fruits. We are accustomed to regard this type of parasitism as primitive, notwithstanding the fact that sometimes the parasites concerned may belong to advanced taxonomic groups; thus *Botrytis cinerea* and *Endostigme inaequalis* belong to the Ascomycetes, and *Corticium vagum* to the Basidiomycetes.



FIG. 71. Branch of *Pinus montana* (mountain pine) attacked by *Herpotrichia nigra* (brown felt blight, 'brauner Schneeschimmel'). Summer aspect. $\times \frac{1}{3}$. (After Gäumann *et al.*, 1934.)

Other pathogens regarded as parasitically advanced show a definite affinity for certain tissues and organs of the host. Their infection germs normally tend to accumulate in the same place, i.e. there is an internal localization of the pathogen and of the disease which amounts to a selective regional colonization by the pathogen. The methods of colonization of the host in the infectious diseases of plants so far studied may be grouped under the following four sub-headings: (a) internal or external to the tissues, (b) tissue specific, (c) organ specific, and (d) organotropic and histotropic.

(a) Internal and External Colonization of the Host

The vast majority of plant pathogens colonize the interior of their hosts; they occur, therefore, inside the tissues, being endophytic or endoparasitic. Some groups of pathogens, e.g. viruses and bacteria, employ no other method of colonization.

Certain parasitic fungi, however, grow either entirely or partially on the outer surface of the host plant, i.e. they are external to the host tissues, growing ectoparasitically. A continuous series may be arranged from facultative ectoparasites to obligate epiphytic parasites.

At one end of this series are forms with an aerial mycelium, whose hyphae can grow freely in the air provided the environment is suitable. They can

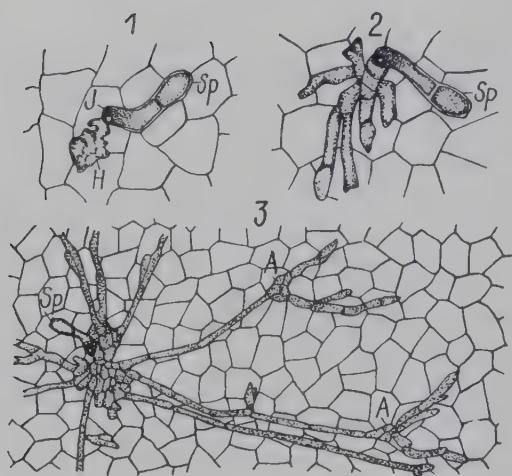


FIG. 72.

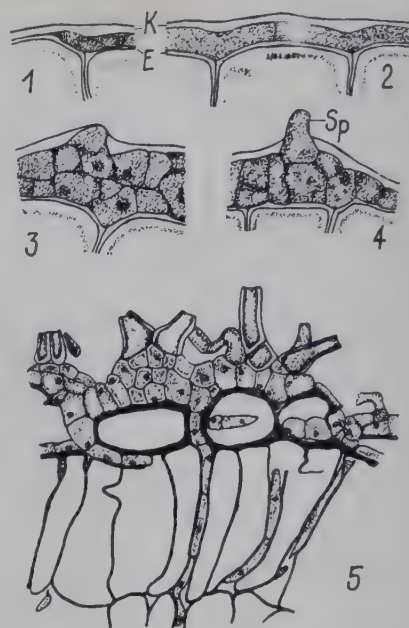


FIG. 73.

FIG. 72. Surface view of the early stages of colonization by *Endostigme inaequalis* (apple scab). 1 The conidium *Sp* has germinated and penetrated the cuticle at *f* with its infection hypha *H*, through a fine microscopically almost invisible pore. 2 Runner-like hyphae spreading radially under the cuticle from the locus of infection. 3 At *A* the hyphae are beginning to form a weft, preliminary to the formation of conidial fructifications (acervuli). 1 and 2 $\times 450$, 3 $\times 200$. (After Nusbaum and Keitt, 1938.)

FIG. 73. Early stages of colonization by *Endostigme inaequalis* (apple scab) in transverse section. 1 A runner hypha advancing between the cuticle *K* and the outer wall of the epidermis *E*. 2 Later stage of a subcuticular hypha. 3 Subcuticular hyphae forming a weft preparatory to forming a conidial fructification. 4 Hyphal branch penetrating the cuticle and beginning to abstrict a conidium *Sp*. 5 Late stage after exfoliation of the cuticle; fungal hyphae growing inter- and intracellularly into the damaged leaf tissues. 1-4 $\times 530$, 5 $\times 310$. (After Nusbaum and Keitt, 1938; and Wilson, 1928.)

thus form a weft round the host plant and continually reinfect it from outside; compare, for example, the brown felt blight (*brauner Schneeschimmel*) of conifers growing under the snow (Fig. 71) and the (white) snow mould of winter cereals (*Fusarium nivale*).

Closer connexion with the host is established by *Corticium vagum* (Fig. 58) whose hyphal weft, growing in loose soil or saturated air, surrounds the bases of potato stems with a white 'crust' and destroys them one after another. Again, in the case of *Ophiobolus graminis*, which causes a foot rot of wheat (Fig. 91), the primary infection does not usually occur at the injured base of the culm but at a more or less distant point of the root system. The fungus grows along the roots by means of runner hyphae, i.e.

externally, until it reaches the culm base where, finally, it begins an endophytic mode of life (secondary infection).

A higher stage of parasitism is represented by the 'cuticle specialists'. Such pathogens grow luxuriantly as ectoparasites between the cuticle and the outer walls of epidermal cells of living leaves, i.e. they are subcuticular. These parasites are able to colonize the internal tissues only on or after the death of the leaves; examples are *Endostigme inaequalis* (apple scab, Figs. 72 and 73), *E. pirina* (pear scab), and *Diplocarpon rosae* (black spot of rose, Figs. 74 and 75). Although the hyphae of these parasites are strictly subcuticular and do not send haustoria into the epidermal cells, they withdraw osmotically from the latter sufficient nutrient material to produce abundant conidial fructifications (the imperfect state) even during the ectoparasitic phase. In contrast, the fruiting bodies of the perfect state, the pseudothecia, are developed internally, i.e. inside the rotting host tissue.

A still higher stage of parasitism has been reached by the Erysiphaceae (powdery mildews) and the tropical Perisporiaceae, which are attached exclusively to the surface of the host and which reproduce only in this position. The mycelium of the Erysiphaceae is external and usually hyaline; it invades the epidermal cells by means of haustoria (Fig. 76) and, more rarely, hyphal branches penetrate into the interior of the leaf (Fig. 77).

Similarly, the higher Perisporiaceae tend towards an epiphyllous mode of life, showing a reduction in the amount of mycelium inside the host tissues and, in so doing, assuming an asteroid habit. Their brown mycelium generally grows over the leaf surface in radiating strands and attaches itself to the leaf by short, mostly unicellular, and often regularly arranged, appressoria-like side branches, termed hyphopodia (Fig. 79). Other side branches, the so-called stomatopodia (Fig. 78), penetrate through the stomata into the mesophyll where they branch and form numerous haustoria, similar to the 'sinkers' of *Phyllactinia*. This asteroid habit is developed so strongly in their commonest genus, *Meliola* (Fig. 79), which parasitizes the leaves, fruits, and, more rarely, the twigs of some 300 species of tropical and subtropical plants, that its representatives have often been regarded as saprophytic epiphytes; they are, however, epiphytic parasites.

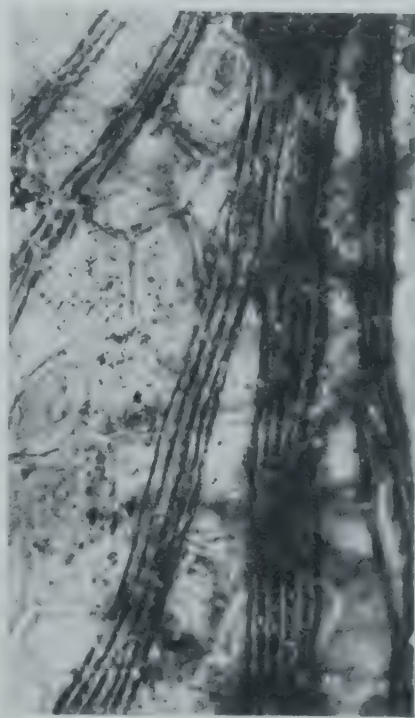


FIG. 74. Parallel strands of runner-like, subcuticular hyphae of *Diplocarpon rosae*, spreading radially on the upper surface of rose leaves. Their dendritic arrangement gives rise to the characteristic clinical picture in black spot disease of rose. $\times 450$. (After Frick, 1943.)

(b) *Colonization of Specific Host Tissues*

Certain endoparasitic pathogens exhibit still further topographic specialization. For reasons which are still obscure, they mainly colonize particular

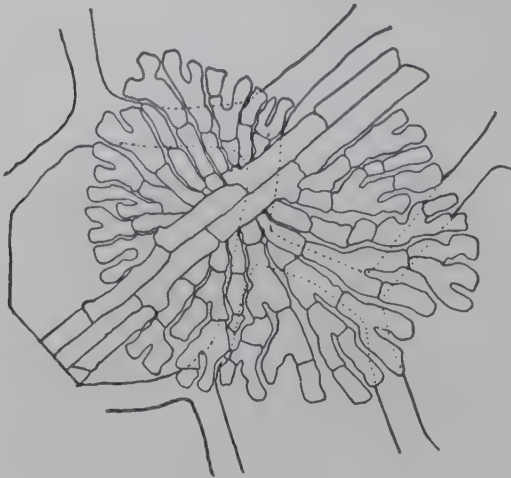


FIG. 75.

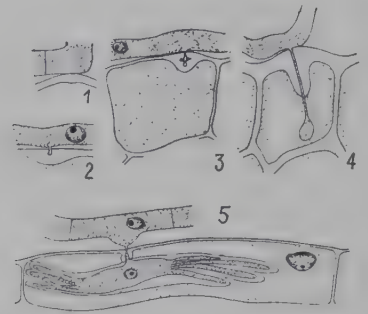


FIG. 76.

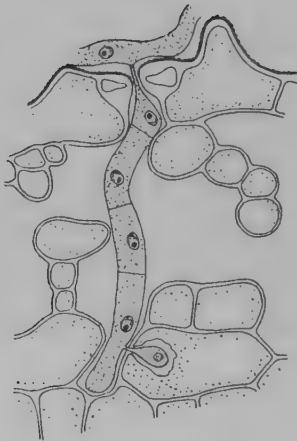


FIG. 77.

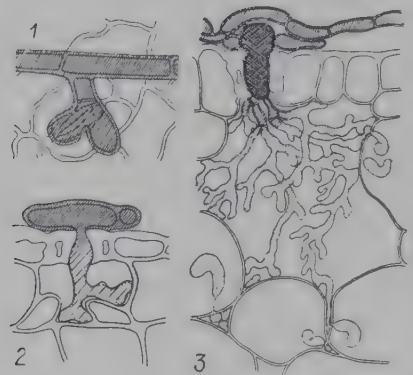


FIG. 78.

FIG. 75. Hyphal branches divided like the fingers of a hand spreading radially from parallel, subcuticular mycelial strands of *Diplocarpon rosae*, thus forming the primordium of a summer conidial fructification (subcuticular acervulus). $\times 700$. (After Frick, 1943.)

FIG. 76. 1-4 Colonization of epidermal cells of *Geranium maculatum* by the powdery mildew, *Sphaerotheca fugax*. 5 Haustorium of *Erysiphe graminis* in an epidermal cell of *Poa pratensis*. $\times 600$. (After Smith, from Gäumann, 1926.)

FIG. 77. Hyphal branch of the powdery mildew *Phyllactinia suffulta* penetrating into the mesophyll of *Cornus stolonifera*. $\times 450$. (After Smith, from Gäumann, 1926.)

FIG. 78. Stages in the colonization of tropical *Perisporiaceae*. 1 and 2 A young stomatopodium of *Parodiopsis Stevensii* seen from above, and in longitudinal section. 3 *Parodiopsis megalospora* in longitudinal section, showing both external and internal mycelium. 1 and 2 $\times 335$, 3 $\times 265$. (After Arnaud from Gäumann, 1926.)

tissues in the host or reproduce themselves in particular tissues, i.e. they show tissue specificity.

Thus, the rust fungi colonize only unligified, assimilatory ground tissue. *Synchytrium endobioticum* (wart disease of potato) and the Erysiphaceae colonize mainly the epidermal cells of leaves and stems. *Pseudopeziza tracheiphila* (Rotbrenner of vine) grows only in the vascular tissues of

leaves. *Phytophthora cambivora* (ink disease of chestnut) attacks primarily the cambium of roots and stems. The central European Hymenomycetes attack only the wood of stems, never green assimilatory tissues; they fall into two classes, one being specific to sap-wood and the other to heart-wood. Thus, *Lenzites heteromorpha* (*Flammung* of timber) destroys the young sap-wood of felled but still living conifers whose bark has not been stripped or which have been left in a sappy condition; conversely, *Trametes pini* (Fig. 295) colonizes only the heart-wood of standing trees.

Some pathogens, such as *Pseudopeziza tracheiphila*, growing inside the host appear almost to 'seek' their specific place of colonization: the initial infection may occur anywhere in the epidermis, whence the young hyphae penetrate into the vessels by a comparatively short route. Here they remain localized but damage the palisade and mesophyll tissues by their toxic excretions in the same manner as those parasites with infection foci. They induce a red coloration of the cell walls, cause disintegration of chloroplasts, &c., and soon lead to premature death of the tissues.

It is only a short step from this high level of tissue specificity to organ specificity (section *c*) and organotropism (section *d*).

(*c*) Colonization of Specific Host Organs

Specific organ colonization includes those cases in which only particular organs of the host are receptive to the pathogen; only in these organs can the pathogen obtain a foothold and undergo further development.

The classic example is the ergot fungus (*Claviceps purpurea*) which lives exclusively in the female sex organs of grasses. Ascospores infect the stigmas at flowering time, and the hyphae grow down into the ovary which they permeate completely, transforming it into a soft, dirty white and wrinkled fungal pseudomorph (Fig. 80, 1). On the surface of the furrows the closely packed hyphal tips cut off masses of hyaline conidia; these are embedded in a sweet fluid which falls in drops from the ear and is known as 'honeydew'. When this type of fructification becomes exhausted the whole body, starting from the base, is progressively transformed into a dark violet, horny sclerotium, the actual ergot (Fig. 81). This shows active intercalary growth, so that eventually it protrudes from the ear. As the host

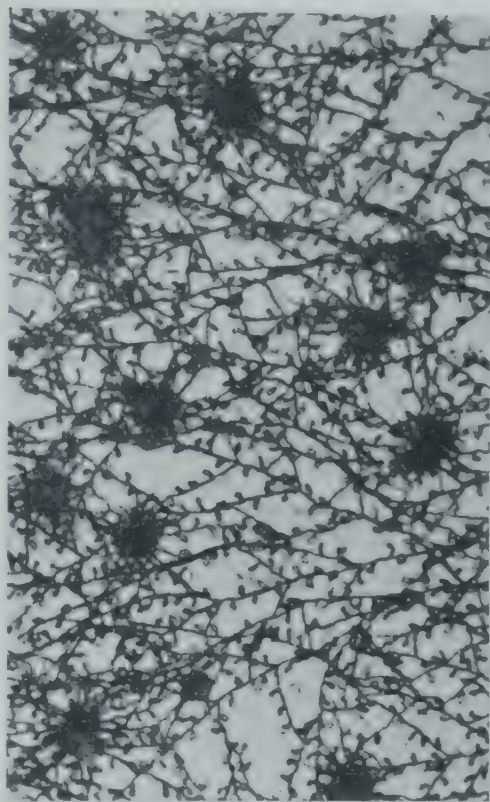


FIG. 79. Epiphytic mycelium of *Meliola guianensis* with fruiting bodies and hyphopodia on cocoa leaves. Approx. 200. (After Stevens and Dowell, 1923.)

maturing, the ergot falls to the ground where it overwinters and germinates early in the following summer, producing its ascogenous fruiting bodies.

Ergot-like bodies found occasionally on the culms or nodes of grasses may seem to argue against so strict an organ specificity in this fungus. They are, however, caused by traumatic infections of the dormant initials of secondary inflorescences which are situated below the sheath and, perhaps,



FIG. 80. Development of the ergot of *Claviceps purpurea*. 1 External appearance of a young ovary of rye; the surface, with the exception of the apex, is covered with the yellowish-white furrowed, conidia-forming mycelium of the parasite; at the apex are the ovarian hairs *H* (the later beard) and the shrivelling stigma. 2 Section of the foregoing; the cavity of the ovary is almost filled with the cavernous, conidia-forming mycelial weft *K*; at the base the primordium *S* of the later sclerotium appears as a small grain. 3 The young sclerotium has developed further and compressed the mycelial weft *K*; at the apex the remains of the ovary *F* are still visible. 4 External appearance of stage 3; at the base the developing sclerotium, in the middle the remains of the conidia-forming mycelial weft, at the apex the 'cap', i.e. the remains of the ovary, style, stigma, and stamens. 5 Semi-mature sclerotium, the later 'ergot' (*Secale cornutum*) of the pharmacist; the conidia-forming hyphal weft can still be recognized by its deep furrows. 1, 2, 4 and 5 approx. $\times 15$, 3 approx. $\times 30$. (After Tulasne, 1853.)

correspond to the axillary and cleistogamous flowers of *Diplachne serotina*. Such stem-borne sclerotia can also be produced experimentally (Fig. 82).

Thus, the ergot really represents a strictly organ specific disease of grasses, infecting only the female sex organs. There are no specific infectious diseases exclusive to the female organs in human pathology; hence specificity in this respect is more marked in infectious plant diseases. In regard to parasitism and sex relations plants are more narrowly specialized than man; in plant diseases, for example, a case of sex specificity occurs in which the female strains of the parasite attack only the male individuals of the host and vice versa, for instance, in the lower fungi (Mucoraceae) the fungus *Parasitella*+ attacks only the fungus *Absidia*—, and *Parasitella*— only *Absidia*+ (Burgeff, 1920).

Similar organ specific relations obtain in the wintergreen rust (*Chrysomyxa pyrolae*) and the bird cherry rust (*Thekopsora areolata*), both of which produce their aecidia only on the scales of female cones of spruce trees (e.g. *Picea excelsa*), that is, presumably on female tissues. Further examples are *Taphrina Johansonii* and *T. alni-incanae*, which produce blister-like growths up to 3 cm. long on the bracts and ovaries of poplars and alders.



FIG. 81.

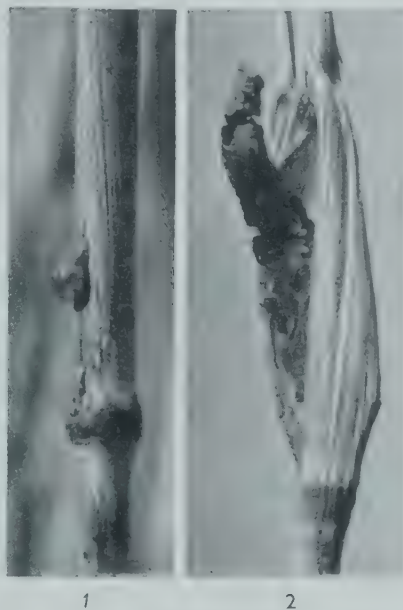


FIG. 82.

FIG. 81. Ear of rye with sclerotia of the ergot fungus (*Claviceps purpurea*). Approx. $\times \frac{2}{3}$. (After Stoll and Brack, 1944.)

FIG. 82. Experimental induction of ergot sclerotia at the uppermost node of a rye stem. 1 Drop of honeydew being exuded, 4 weeks after infection. 2 Ergot sclerotium borne on the stem. Approx. nat. size. (After Stoll and Brack, 1944.)

A certain tendency towards organ specific colonization of the host is present in the fruit tree Sclerotinias: *Sclerotinia Linhartiana* on quinces, *Sclerotinia fructigena* on various pome fruits (Fig. 112), *Sclerotinia laxa* on apricots, and *Sclerotinia cinerea* on other stone fruits. These fungi also attack the leaves and twigs of their hosts as well as the fruits. On the leaves and twigs they produce only their imperfect reproductive state, the conidia, i.e. the leaves and twigs serve as subsidiary host organs. The perfect reproductive state, the apothecia, are produced only on the fruits of the host, i.e. the fruits serve as principal host organs. Different host organs, therefore, have different biological values for the pathogen.

Finally, the rust fungi show a negative organ specificity. They are unable to attack the microsporophylls (stamens) and their derivatives, the petals of flowering plants. In addition, however, to foliage leaves and

stems, they can also attack the macrosporophylls (carpels, ovaries), and perigone leaves, e.g. sepals (modified bracts).

(d) Organotropic and Histotropic Colonization of the Host

In a number of infectious diseases of plants generalization leads at once to general infection: the pathogen sweeps through or permeates the entire host organism. During the further course of the infection, however, the disease germs tend to aggregate in one predetermined locality, in certain organs or tissues. Here they ultimately establish themselves and attain their optimum development and, in mycoses, produce their fructifications.

Consequently, general infection represents only a transitional stage with these pathogens; they spread as it were through the entire host organism until they reach the locality best suited to their further development. They are termed organotropic or histotropic according to whether they show special affinity for particular organs or for particular tissues.

Does the host force the parasite to occupy these special regions because they are places of least resistance, or do the pathogens themselves 'select' just these tissues of the host? As yet we cannot answer these questions but it seems likely that in infectious plant diseases the latter hypothesis may be the more correct. Thus, in so far as the parasites concerned can be cultivated at all on artificial substrates, they do not form their sexual or perfect fructifications. For this development to occur, certain narrowly defined conditions evidently need to be satisfied, requirements which are only fulfilled at the 'chosen' localities.

Because of this specific affinity for certain organs or tissues, general infections do not cause a general malady in this group of diseases. Apart from the selected special tissues the parasite exerts little pathogenic effect on the host tissues, evoking in them at most certain non-specific and usually vague general symptoms. The systemic infection is thus passed through as a latent, transitory stage with practically no symptoms.

The actual symptoms become manifest, therefore, in the predetermined 'selected' organs or tissues, i.e. the characteristic features of the disease appear only after such localization has occurred. A general infection of the entire host organism is, therefore, followed by a local outbreak of the disease (localization of lesions). Hence, such a disease is termed cyclic, its course invariably passing from initial infection through generalization to local manifestation. This contrasts with the usual course in which infection leads through generalization to general disease.

In human pathology many important infectious diseases conform to this cyclic scheme: first, infection through the mouth cavity; next, generalization through the circulatory system, with general symptoms, e.g. rigors; and later local manifestations, for example, organotropic fixation in the lungs (croupous or lobar pneumonia), or histotropic fixation in the nervous system (neurotropic viruses, e.g. poliomyelitis), in the cerebral membranes (meningococci), or in the skin (dermatotropic viruses, e.g. measles, German measles, and smallpox).

By contrast, such cyclic infectious diseases are rare in plant pathology. This may be related to the fact that the differentiation of organs and tissues in the plant is of a lower order than that in the human body.

The parasites discussed on page 50 provide examples of organotropic pathogens, which make their way through seedlings, e.g. bunt of wheat, anther smut of the Caryophyllaceae, and the loose smuts of oats, wheat, and barley (Fig. 98). All these parasites normally attack their hosts when these are in their earliest stages and invade the growing-points. They grow systemically through all the shoots, leaves, &c., causing diffuse general symptoms, but finally becoming localized in the region of the flower; thus, bunt of wheat appears in the female organs, the anther smut in the male organs, and the loose smuts in all primordia of the flower.

There is no necessity, however, for such general infections to produce local manifestations in specific organs. If, for instance, barley plants attacked by *Ustilago nuda* (loose smut) are prevented artificially from shooting, then the fungus produces its sori in the foliage leaves instead (Lang, 1913). This internal regulation still remains a puzzle because quite different hyphae of the parasite are concerned in spore formation in the leaves and in the ovaries respectively. The medical man might say in analogous cases that the host organism normally forces the parasite towards the place of least resistance, in this case towards the flowers. Suppression of shooting would then deprive the parasite of its 'predilected' site, hence the disease breaks out in a non-specific site. It is, however, not really possible to imagine such a course of events in the plant body, which forms so much less an integrated whole than the human body.

Functionally, it is to be noted that in all these examples the 'forcing of the parasite', or however one chooses to interpret the phenomenon, occurs in the direction of the sex organs of the host, while in man it is just this form of localization that is absent. However, in the plant the sex organs are practically the only clearly individualized organs of the body. If one uses the expression 'venereal diseases' in order to elaborate the contrast with human pathology, then three types are found in the plant kingdom:

1. Venereal diseases in the medical sense. In this case the sex organs represent only the elective path of infection, from which the pathogen invades the entire host organism. Examples are *Bacillus amylovorus* (Fig. 15) and the *Monilia* disease of twigs.
2. Venereal diseases specific to the sex organs. Here the sex organs alone are attacked and become diseased; the classic example is *Claviceps purpurea* on the female sex organs of grasses. This type is entirely lacking in human pathology.
3. Organotropic venereal diseases, where, as was mentioned just now, a systemic infection regularly ends with a local manifestation in the sex organs and in them alone. This type is also lacking in human pathology.

Leaf stripe disease of barley, leaf smut of bistort, and phloem necrosis of potato are examples of histotropic pathogens.

The mycelium of *Helminthosporium gramineum* (leaf stripe of barley) grows systemically through the whole plant body right from the seedling stage (Fig. 90); but its reproductive bodies are produced mainly on the leaves, where the longitudinal stripes form characteristic lesions, hence the name. The same occurs in the stripe or flag smuts of wheat (*Urocystis tritici*) and of grasses (*Ustilago striaeformis* and *U. longissima*).

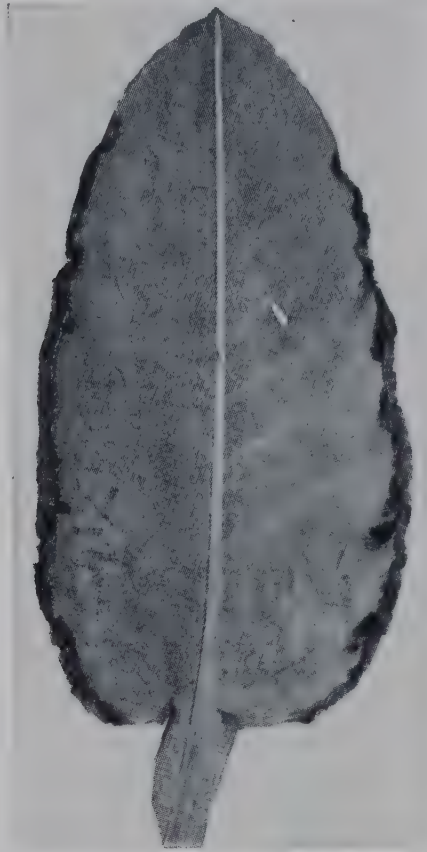


FIG. 83. Sori of spores of *Ustilago marginalis* borne on the margin of a leaf of *Polygonum Bistorta*, an example of histotropic colonization of the host. Nat. size. Orig.

Ustilago marginalis (leaf smut of bistort, *Polygonum Bistorta*) is perennial in the rhizome. Each year it permeates all the shoots and leaves, but its reproductive bodies are produced only along the edges of the leaves which appear as if in mourning (Fig. 83).

The viruses causing leaf roll of potato and curly top of sugar beet pervade the entire plant body, but establish themselves mainly in the phloem where they cause specific injuries (phloem necrosis, Fig. 281).

The reasons for such characteristic localizations have still to be discovered; why, for instance, among all the tissues available, should *Ustilago marginalis* 'select' the edges of leaves for its most intensive development.

CHAPTER 2

INFECTION CHAINS

How does a pathogen gain access to a host? In a manual this process would be discussed in the first chapter because, logically and in order of time, it precedes infection. On the other hand, in a textbook, the description of infection must come first because it embodies a series of fundamental conceptions.

An infectious disease can maintain itself only by continual reinfections on the basis of infection chains and if these are interrupted it dies out. By an infection chain is meant the continuous, serial transmission of infective material from host to host. The infected host is termed the distributor, the plant to be infected the receiver, and the individual links in the infection chain are termed generations.

In regard to the continuity of the links and the way in which they are arranged the infection chains show differences in their construction; hence § 1: 'The Constitution of Infection Chains.'

The chains originate in a source of infection; hence § 2: 'The Sources of Infection' (p. 89): where do the disease germs maintain themselves outside the host?

From the source of infection the disease germs somehow reach the host. The ways in which they are spread are described in § 3: 'The Transmission of Pathogens' (p. 106).

And finally, the question arises, what conditions are necessary for infection chains to lead to cumulative outbreaks of disease: § 4: 'The Epidemiology of Infectious Plant Diseases' (p. 134).

§ 1. The Constitution of Infection Chains

In regard to their continuity infection chains may show interrupted or uninterrupted progress.

Continuous infection chains invariably lead without a break directly from one host to another. They are familiar in human medicine, for example, in measles, scarlet fever, and diphtheria. Here, the continuity of the infection chain is a vital necessity for the pathogen, since it forms neither spores nor other resting stages, nor can it maintain itself saprophytically apart from a host; it succumbs, therefore, unless continuously provided with fresh hosts to infect.

Among the infectious plant diseases of the temperate zone only the viroses, e.g. potato mosaic (Fig. 148), depend entirely on continuous infection chains of this kind. Intermittent infection chains are much more usual in our vegetation zone where a winter season, during which hosts are unavailable, temporarily interrupts the serial transmission of the pathogen from one host to the next; in such cases, where there is no suitable host link,

the gap is bridged by a saprophytic phase or a resting state, e.g. spores or comparable elements. Moreover, the phytopathogenic bacteria which, as has been stated, do not produce spores and might, therefore, be expected to form continuous chains analogous to those of diphtheria, develop discontinuously in our vegetation zone and, for a certain period between the parasitic phases, live saprophytically in the soil.

In regard to the arrangement of their links, infection chains may be homogeneous or heterogeneous (Doerr, 1941, 1942).

In homogeneous infection chains the disease germ or contagion is always transmitted to hosts of the same species or species-group, e.g. from potato plant to potato plant or from apple tree to apple tree; the parasites in question are confined to one host species, i.e. they are autoecious. Heterogeneous infection chains, on the contrary, involve hosts belonging to different species; the parasites alternate between different hosts, i.e. they are heteroecious.

(a) *Homogeneous Infection Chains (Autoecious Pathogens)*

In plant pathology most infection chains are homogeneous. Apple scab (*Endostigme inaequalis*) may serve as an example. Typically, it overwinters in rotting apple leaves on the ground; these contain its reproductive primordia from which the sexual reproductive state, the ascospores, develop in the spring and infect the young foliage leaves. During the summer and autumn it spreads by asexual propagative spores, the conidia (Fig. 73), in a long chain of generations from leaf to leaf, and thence to the young branches and the fruits (Fig. 86) until the autumn, when the close of the growing season ends its development for the current year and initiates the production of the sexual or perfect state.

Thus, it is the systematic relationship of the host and not the spore form that decides the homogeneity of the infection chain; the pathogen must parasitize exclusively one and the same host species or species-group. The spore form is decisive only as regards the continuity of the infection chain; the conidia prolong the continuous phase, whereas the ascogenous fructifications start the discontinuous phase. Hence, as in most plant diseases, the apple scab infection chain is homogeneous but discontinuous.

Within the rhythmic developmental sequence of the several organs of a host or of the various principal and subsidiary hosts, repeated variations may occur every year throughout the growing period in the sequence of the host organs or hosts to be colonized by the homogeneous infection chain.

In the simplest case this variation may be merely a change of organ on the one host, as in the case of apple scab which attacks the foliage leaves at the opening of the growing period and the young fruits later; the same applies to the *Sclerotinia* spp. on fruit trees mentioned on page 77.

Where the order of appearance of susceptible organs involves different host species a change of organ necessitates a change of host. For instance, in *Sclerotinia rhododendri* on alpenrose, the mummified fruits fall to the ground in the autumn where they overwinter and, in May, produce the

cup-shaped apothecia, i.e. the perfect fructification. The ejected ascospores, however, are unable to attack alpenrose foliage; they infect the young unfolding leaves of bilberry (*Vaccinium Myrtillus*) and bog whortleberry (*V. uliginosum*), two secondary hosts on which the pathogen grows only until the development of the imperfect or conidial state. During the summer the conidia infect the flowers of alpenrose, their germ tubes penetrate through the stigma and the stylar canal into the ovary below, and convert the fruit into a mummy or sclerotium. This falls to the ground and, in the following spring, once more proceeds to the inception of the perfect state, the apothecia.

Hence, from the standpoint of the parasite, the change of hosts by *Sclerotinia rhododendri* is purely facultative, being determined not by the constitutional development of the pathogen, but by the varying rhythm of development of the several vicarious hosts; the latter, moreover, unlike those of the rusts to be discussed shortly, are closely interrelated, belonging to the same family.

In still other cases the developmental rhythm of the same organ, e.g. the time of flowering, varies in the different host species so that a regular succession of links develops within the homogeneous infection chain. *Claviceps purpurea* f. sp. *brachypodii* Stäger, a specialized race of ergot (Fig. 81), may serve as an example.

The sclerotia formed on the slender false brome grass (*Brachypodium sylvaticum*) germinate in April or May at which time, however, the panicles of this grass are still deeply enveloped in the leaf sheaths and are not accessible to infection of any kind. In its place the wood millet grass (*Milium effusum*) is available in its flowering stage. The ascospores infect the stigmas of this grass and for several weeks the mycelium produces masses of conidia (honeydew) on the ovaries but, strangely enough, rarely or never produces normal sclerotia. *Milium effusum* is only a subsidiary host but, nevertheless, the infection progresses continuously from flower to flower for 2–3 months. At the end of July or beginning of August *Brachypodium sylvaticum* comes into flower and is infected by conidia from *Milium effusum*. Only on the former, the principal host, does the parasite achieve its full development and produce sclerotia. Ergot on *Brachypodium* can, therefore, continue to exist only where this grass is associated with *Milium effusum*. Thus, the chain originating in the sclerotia, the intermittent source of infection, is perpetuated through an ancillary host, the wood millet grass (transitional infection), until the principal host, slender false brome grass, has reached the susceptible stage of development. Here, too, as in *Sclerotinia rhododendri*, the change of host, considered from the standpoint of the pathogen, is facultative and the substitutive hosts are closely interrelated, again belonging to the same family.

(b) *Heterogeneous Infection Chains (Heteroecious Pathogens)*

In heterogeneous infection chains (p. 82) the sequence of generations is interrupted, as a result of innate tendencies, by a transition to a host

belonging to another species. This transition may, up to a certain point, be facultative but, on the other hand, it may be obligate, i.e. it may involve regular alternation.

(aa) Facultative heterogeneous infection chains. The infection chain in black rust of cereals (*Puccinia graminis*) is facultatively heterogeneous. The teleutospores overwinter on the dry culms and leaves of the grass and germinate in the spring producing basidia in which the reduction division takes place. The haploid basidiospores arising on the basidia are not able to infect the teleutospore hosts (Gramineae) on which they were produced, their aggressiveness being confined to the young leaves of Berberidaceae, systematically an entirely unrelated family; in our country the European barberry (*Berberis vulgaris*) is mainly concerned. In this rust, therefore, the reduction division which restores the haploid chromosome number involves a change in the selection of host: whereas the dikaryophase inhabited the Gramineae, the haplophase can subsist only on the barberry.

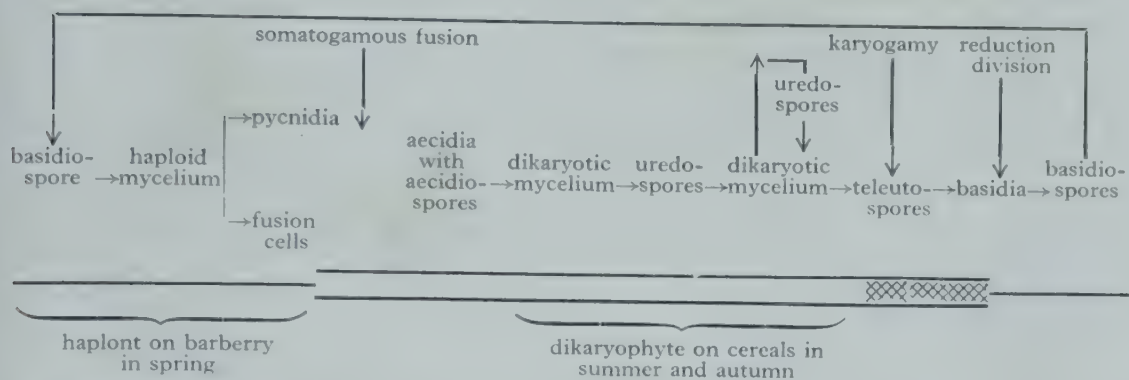
On the upper leaf-surface of the barberry the haploid mycelium forms pycnidia and on the lower one aecidia; here sexual fusions occur resulting in the genesis of the dikaryotic or binuclear aecidiospores. These, in their turn, are unable to colonize the aecidial host, i.e. the barberry, on which they were produced, their parasitic faculty being restricted to the leaves of Gramineae. Hence, in *Puccinia graminis*, sexual fusion giving duplication of nuclei is followed by a regression in host 'choice': whereas the haplophase inhabited Berberidaceae, the newly produced dikaryophase must continue its development on Gramineae, whence the fungus started.

The aecidiospores germinate on Gramineae producing a dikaryotic mycelium in the tissues. This mycelium at first gives rise to asexual conidia, or uredospores, which belong like the aecidiospores to the binuclear phase and are similarly capable of attacking the same host, the Gramineae; accordingly they propagate black rust from one member of this family to another. The production of uredospores involves a disturbance in the equilibrium between the haplophase and the dikaryophase. The former, as we have seen, cannot spread from one member of the Berberidaceae to another since its sole pathogenic spore form, the aecidiospores, are compelled by their binuclear state to effect a change of host; in the absence of the alternate host (in this case the Gramineae), the parasite dies out. The haplophase change of host is, therefore, obligatory. On the other hand, the dikaryophase is able, in so far as climatic conditions permit, to subsist on the Gramineae independently of the barberry since, by means of its uredospores, it can pass from one member of the Gramineae to another for an indefinite number of generations. The absence of an alternate host, therefore, does not necessarily involve the extinction of the dikaryophase and change of host in the dikaryophase is thus facultative.

Towards autumn the dikaryotic mycelium, which in the intervening period formed only uredospores, now gives rise to teleutospores, i.e. the actual resting spores whereby the black rust overwinters on the dry culms and leaves. Within them the fusion of the nuclei takes place (karyogamy),

and, as explained above, they germinate in the spring by means of a basidium; at this stage the coincident reduction division once more necessitates change of host, namely, reversion to the Berberidaceae, the original host of the haplophase.

The infection chain of *Puccinia graminis* may thus be represented by the following diagram.



By means of the resting spores it progresses from cereals to barberry and then by means of the aecidiospores back again to cereals and through the uredospores from cereals to cereals . . . cereals . . . cereals . . . and so on, finally reverting by means of the resting spores to barberry. It is thus discontinuous, because the stage of development of the host or the climatic conditions induce the formation of the resting spores whereby the pathogen bridges the months during which the host is lacking. It is heterogeneous, because the resting spores must be followed by a new host, barberry, and it is facultatively heterogeneous because this change of host applies only to the haplophase and not to the dikaryophase, in which, on the contrary, an indefinite number of generations may be formed successively on individuals of the same species.

Since, in this group of examples, the change in the nuclear phase of the fungus is responsible for the heterogeneity of the infection chain, the latter can be rendered homogeneous if the nuclear change be prevented. Thus, *Cronartium asclepiadeum* (stem blister rust of pine, Fig. 52) is normally compelled to migrate from pine (the aecidial host corresponding to the barberry) to white swallow wort, *Vincetoxicum officinale*, &c. (the teleuto-spore host corresponding to cereals). Should somatogamous fusion fail to take place because of growth disturbances in the aecidia, the morphological rhythm of development may still proceed in its absence but apomictically and, in such cases, the aecidiospores are uninucleate; this type of behaviour has been established cytologically for some other rusts (Gäumann, 1926, p. 428). These uninucleate aecidiospores again attack pines so that, as the forester Haack assumed more than a century ago on the basis of practical observations, the infection chain proceeds as a homogeneous pathogen from pine to pine even in the absence of the alternate host which, in this case, is no longer necessary. The infection chain is homogenized.

(bb) Obligate heterogeneous infection chains. In obligate heterogeneous

discolorations of the foliage, accompanied by inward rolling or folding of the leaves and leaflets which gives the disease its name. The virus cannot be transmitted to new potato plants by laboratory techniques, such as the inoculation of cell sap, but must pass through the body of certain specific aphides, e.g. the peach leaf aphis (*Myzodes persicae*) or an allied species (vector transmission), and then again be introduced by the insects into the phloem of the plants to be infected (Fig. 85). It is, however, transmissible directly from the mother plant to the tubers whence it again penetrates into the young plants; in the same way it migrates through graft unions where its passage is arrested only by some inanimate medium such as a knife, a necrosed wound surface, &c.

Two features are characteristic of this mode of development:

1. The obligate nature of the host change. In the transmission of the virus from one potato plant to another, the two links of the infection chain, the potato plant and the peach leaf aphis (like the pear and the savin in the cluster cup rust) alternate regularly: potato plant—leaf aphis—potato plant—leaf aphis—potato plant. On the other hand, the infection chain does not pass directly from one potato plant to another; like the cluster cup rust it proceeds continuously because no resting period is interpolated, and it is obligatorily heterogeneous since each nuclear change is associated with host change. In the leaf roll disease, however, the alternating host, the insect, in contradistinction to the savin, is not merely an alternate host but also a vector like *Anopheles* in human malaria.

2. The specific character of the alternate host. The leaf roll virus, for reasons as yet unknown, is transmissible only by certain insects, although the anatomical structure and mode of life of others would seem to qualify them equally well; the virus may, however, undergo a specific reorientation of development in the specific vector.

The route and the location of the leaf roll virus inside the alternate host, the insect, are known only approximately. In other viroses of the same group, e.g. leaf curl of sugar beet and maize streak, the virus seems to pass from the alimentary canal to the blood stream and thence to the salivary glands reaching the plant to be infected with the saliva during feeding.

The insect, however, cannot effectively transmit the virus directly after feeding; it is not contagious immediately after sucking a diseased plant but

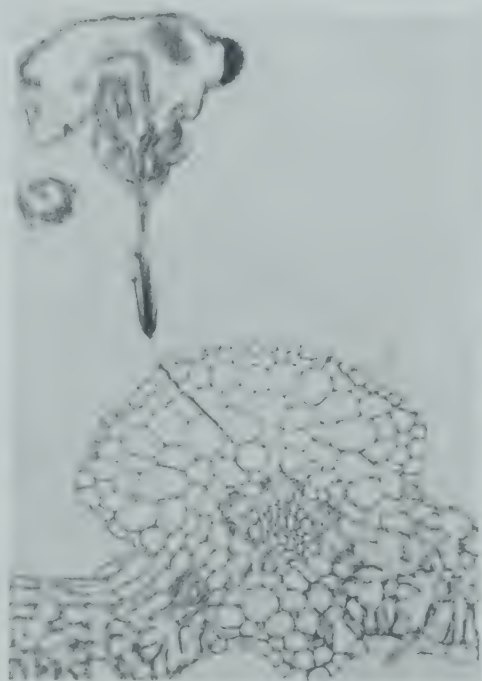


FIG. 85. Section through a potato leaf from whose phloem an individual of *Myzodes pseudosolani* is withdrawing nutriment. $\times 55$. (After Dykstra and Whitaker, 1938.)

only after a certain lapse of time, during which it can feed at will on healthy plants without infecting them. This interval is sometimes termed the 'incubation period' but the expression 'circulation period' is preferable because it avoids any confusion with the true incubation period on the potato plant. In the case of the leaf roll virus the circulation period ranges from 12 to 48 hours; in leaf curl of sugar beet, transmitted by the leafhopper *Eutettix tenellus*, it is not less than 4 hours; and in aster yellows, transmitted by another leafhopper, *Cicadula sexnotata*, it is at least 10 days. Its duration does not seem to be influenced by the amount of virus absorbed but high temperatures appear to shorten it.

It has been assumed that the virus multiplies in the insect's body but the relevant observations mostly admit of other interpretations. At all events, it is established that certain insects, provided they have taken up enough of the virus, remain infectious all their lives. In only one instance, that of aster yellows, has convincing evidence been furnished (Black, 1941) that the virus multiplies at least an hundredfold between the second and the tenth day of the 17-day circulation period.

We are still completely ignorant regarding the biological significance of this form of host change, and of the reason why certain viruses must pass through specific insects as alternate hosts before they again become infectious.

The existence and mechanism of heterogeneous infection chains confront biologists with interesting problems, since three organisms, a host, an alternate host, and a micro-organism, have been constrained to unite in a biological community with its own laws.

On the spatial plane a biological community of this kind presupposes the occurrence of the two alternating hosts in the same vicinity; sociologically, they must belong to the same or adjacent plant units (plant societies, life zones, and so forth). This applies particularly to parasites which in respect of their alternate host are monophagous, like the cluster cup rust of pear trees, whereas the sociological affinities of polyphagous parasites such as *Cronartium asclepiadeum*, the bark rust (resin top) of Scots pine, which in addition to *Vincetoxicum officinale* also makes use of *Paeonia officinalis*, *Pedicularis palustris*, &c., as alternate hosts, are not necessarily so restricted.

Moreover, heterogeneous infection chains are, in general, limited to the biological community to which both hosts belong; a few exceptions due to human intervention will be discussed in connexion with the epidemiology of plant diseases. Thus, the heteroecious rusts are tied to certain plant societies within which they circulate and persist (Baxter and Wadsworth, 1939). This explains the wealth of such rusts in areas of relict flora.

Finally, there are the problems of the phylogenetic development and geological age of heterogeneous infection chains. Little is known of their phylogenetic development and, with regard to their geological age, we are entirely dependent on inference. The heteroecism of many rusts probably dates back far into the Mesozoic Period (Dietel, 1918). Further, the types of modern heteroecism occurring in geographically disconnected

areas, such as the Alpine and Circumpolar regions, must certainly have been in existence before the Ice Age, prior to the breaking-up of their originally continuous area (Fischer and Gäumann, 1929).

§ 2. The Sources of Infection

Where does the pathogen live when outside the host? In infectious plant diseases the chief reservoirs, providing a constant renewal of infection chains, are: (a) an infected host; (b) saprophytic stages in the life cycle of the pathogen outside the host, for example, in dead organs of the latter



FIG. 86.

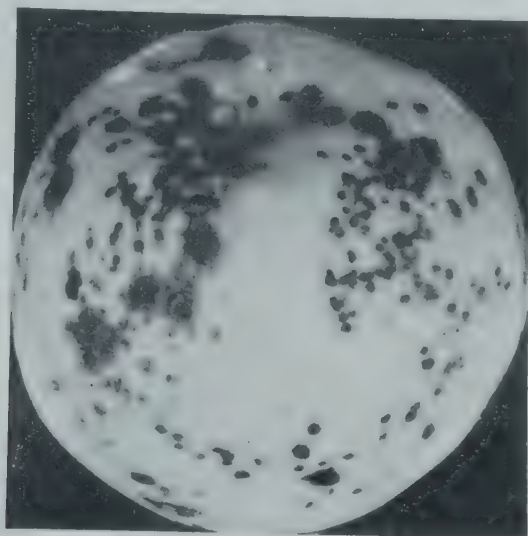


FIG. 87.

FIG. 86. Early scab (May and June) on a young apple of var. Gravensteiner. Approx. $\times 3$. (Original at the Federal Research Institute, Wädenswil.)

FIG. 87. The large, cracked, and scarred lesions of early scab (Fig. 86) have shed spores which have given rise to the still small, disparate foci of late scab. Nat. size. (Original at the Federal Research Institute, Wädenswil.)

or in the soil; and (c) resting spores or other resting stages of the parasite. Thus, in plant disease, there is not the reservoir formation in warm-blooded animals familiar in human medicine, for example, in plague and spotted fever, where the reservoirs are rodents from which the contagion is transmitted by insect vectors to man.

The first link of the infection chain, which originates directly from the reservoir, i.e. in a temperate climate mostly from the overwintering focus, is called the primary infection, and the following ones the secondary infections. For example, in ergot (p. 75), ascospore infections are invariably primary and honeydew infections are secondary; in the cereal fusarioses (p. 72) seedling infections are mostly primary whereas grain infections are always secondary.

However, in addition to their use in an epidemiological sense, these terms are also applied to differentiate continuing successive infections from disparate foci on the same organ; thus, in apple scab (p. 82), not only the first spring infection of the foliage, arising from the overwintering

focus, is termed primary, but also the first early summer attack on the fruits (early scab, Fig. 86). In this case the secondary infections are the scabs which appear later on the maturing fruits (late scab, Figs. 87 and 88), and the tertiary infections are the economically important lesions developing after the crop is harvested (storage scab, Fig. 89).



FIG. 88. Late scab (summer scab) on var. Landsberger Reinette. $\times \frac{3}{4}$.
(Original at the Federal Research Institute, Wädenswil.)

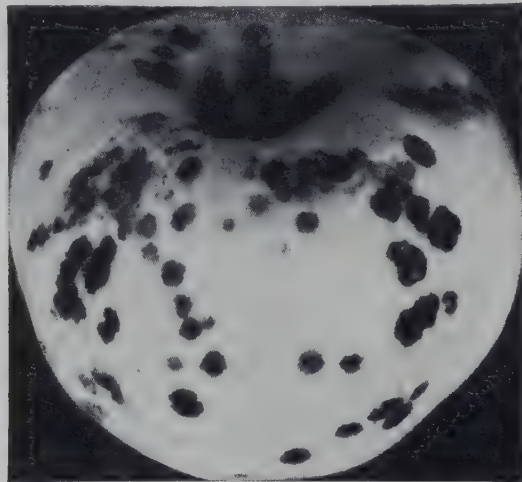


FIG. 89. Storage scab on var. Boiken first appearing during storage in the cellar. $\times \frac{3}{4}$. (Original at the Federal Research Institute, Wädenswil.)

Corresponding to the three chief modes of subsistence of plant pathogenic parasites outside the host mentioned above, the following section is divided into three subsections: (1) the infected host; (2) saprophytic phases in the development of the pathogen; and (3) resting stages of the pathogen as infection reservoirs.

1. *The Infected Host as a Source of Infection*

In plant pathology the distributor is, as a rule, not only infected but also diseased. If, however, it harbours a pathogen without showing any symptoms it is termed a carrier; thus the black nightshade (*Solanum nigrum*) is

a symptomless carrier of several potato viruses, whilst cultivated plants which are tolerant of certain viruses can also serve as carriers.

In the floristic regions of the temperate zone the infected host is most important as a source of infection when it shelters the pathogen over the winter, thereby preserving it during the barren period. Current usage differentiates here between overwintering in the strict sense and perennating, although the two forms of subsistence overlap.

Simple overwintering by means of living mycelium is often effected in our climate on winter annuals. For example, *Puccinia glumarum* (yellow rust of cereals) usually forms no teleutospores in damp climates; its aecidial stage is unknown and in central and northern Europe plays no part in the survival of the species. In these regions the fungus preserves itself by its dikaryotic mycelium which lives from one growing season to the next inside the winter-hardy leaves of autumn-sown cereals and wild grasses. On these leaves, immediately they unfold, the fungus again forms uredospores which propagate it during the summer in a succession of new generations in the cereal fields. The young winter crops, in which the mycelium tides over, are infected in the autumn and so the infection chain is carried forward into the next year.

The same mode of survival in winter crops by means of the mycelium, which after the melting of the snow again proceeds to the formation of asexual spores or oidia, is found in the powdery mildew of cereals, especially *Erysiphe graminis hordei* on barley. Because of the early infection of the neighbouring summer barley fields the mildew epidemics in Denmark, for instance, assumed such proportions as to compel a reduction of winter barley cultivation.

Overwintering foci may be localized in tubers instead of in winter-hardy leaves. For example, in potato blight (*Phytophthora infestans*) the resting spores (oospores) play practically no part in the overwintering which is accomplished, whenever feasible, in the vegetative system of the plant (Fig. 271). The agent of bacterial ring rot of potato (Fig. 62) also perennates in this way.

Other pathogens overwinter in localized infection foci on branches. Thus, in certain regions apple scab (*Endostigme inaequalis*) overwinters in this way producing 'scabby' branches whence the infection chain passes to the young leaves and from them to the fruits.

In still other pathogens, e.g. *Microsphaera alphitoides* (oak mildew), *Sphaerotheca pannosa* (rose mildew), *S. mors-uvae* (American gooseberry mildew), *Podosphaera leucotricha* (apple mildew), and *Uncinula necator* (powdery mildew of grape vine), the mycelium often overwinters on the bud scales and, in the following spring, attacks the green shoot below the bud and the outer leaves during the unfolding of the bud where it produces its first oidia, and so continues the infection chain.

All these modes of overwintering are more or less facultative. Thus, the powdery mildews and apple scab can also survive the winter without the co-operation of their hosts by means of their sexual perfect state, the

perithecia and pseudothecia. From the epidemiological standpoint, however, vegetative overwintering inside the host is by far the most effective way. With the onset of favourable conditions such fungi begin to form spores which give rise to early infections and so permit the development of a correspondingly long sequence of generations in the course of the growing season. The efficiency of vegetative overwintering may be assessed particularly well in the case of *Microsphaera alphitoides*. From 1907 onwards this fungus spread through the whole of Europe and western Asia. Its perithecia were not recorded until 1911 and since then have only rarely been found; the overwintering of the fungus is thus effected exclusively by means of the vegetative mycelium.

As examples of perennation in the strict sense of the term may be instanced the dikaryotic mycelium of the cluster cup rust on the savin (Fig. 84), or *Uromyces pisi* (Fig. 51), whose mycelium overwinters in the rhizome of the cypress spurge, colonizes the buds on the young rhizome, and then permeates the shoots systemically. Also *Melampsorella caryophyllacearum* (witches' broom of silver fir, Fig. 230) perennates for decades by means of its haploid mycelium in the witches' brooms and, by means of its dikaryotic mycelium, for comparable periods in the infected shoots of many Caryophyllaceae, producing external spores anew each season.

Mycelial perennation is also found in *Rosellinia quercina* (root rot of oak). This fungus can overwinter by means of ascospores and sclerotia on or in the soil but it is most formidable when the mycelium persists in the root systems of older trees which serve as symptomless carriers. If a forester establishes nurseries or plantations adjacent to such old stands, because new growth usually thrives best where the particular species is indigenous, the fungus emerges from its locus of chronic infection and decimates the saplings. From an epidemiological standpoint the hosts which harbour a perennating pathogen play the same role as carriers in human medicine.

These parasitically perennating infections are most unwelcome when they lead to the formation of reservoirs of infection on non-specific or subsidiary hosts, whence the parasite constantly re-attacks the principal host. The number of hosts of any practical importance is usually less than the number of potential hosts; hence, control measures are apt to be directed solely towards the eradication of the pathogen on the main host, secondary hosts being neglected, although it is just these that constitute the permanent overwintering and infection reservoirs.

Thus, carriers of wart disease of potato among the wild Solanaceae are an easily overlooked perennial reservoir of disease infection for neighbouring potato fields. Similarly, the numerous wild plants susceptible to bacterial rot (*Bacterium solanacearum*) serve as a perennating source of infection for tobacco plantations (Smith, 1939) so that the disease cannot be eliminated by crop rotation or fallow. Lucerne (*Medicago sativa*) and red clover are commonly attacked in leys by *Rhizoctonia crocorum* (*Rhizoctonia violacea*) but sustain little damage although becoming infection reservoirs; if the field is ploughed up, however, the fungus develops in epidemic form

on the sugar beets or potatoes planted as the first crop in the new rotation. In the same way, lady's smock (*Cardamine pratensis*) serves as a perennating reservoir of infection for *Plasmodiophora brassicae* (club root, Fig. 128), which passes over to rape (*Brassica Napus*) and turnip (*Brassica Rapa*) and may give rise to a serious outbreak on the ploughing up of the meadow.

2. *Saprophytic Phases of the Pathogen as Infection Reservoirs*

The pathogens to be considered here are not dependent upon a strict continuity of infection chains since, if these be interrupted, they can continue their growth as facultative saprophytes and produce accessory reproductive bodies. Hence their saprophytic infection reservoirs serve not only to maintain them in a vegetative state but also to propagate and spread them outside the host. This extended development of the pathogen outside the host differentiates the saprophytic infection reservoirs from the resting spores, &c. (to be discussed in the following section), which remain passive and inactive during the period when a host is lacking.

As in the case of human pathogens, the external saprophytic growth phase in plant pathogenic organisms may occur either (a) on the surface of the future hosts, or (b) in the soil, &c.

(a) *Saprophytic Phases on the Surface of the Future Host as Sources of Infection*

Just as a human being continuously harbours a specific skin flora, of which some elements, e.g. staphylococci and streptococci, may become troublesome as agents of wound suppuration, so plants always harbour an epiphytic bacterial flora of characteristic composition (Burri, 1903), of which at least one constituent, *Bacterium herbicola aureum* (Düggeli, 1904), may be deemed a typical member. This epiphytic bacterial flora of plants is, however, perfectly innocuous and, unlike the corresponding organisms in man, cannot introduce infections even through wounds.

In phytopathology, therefore, the problem of saprophytic infection foci on the surface of the future host arises only in special cases grouped under the somewhat inapposite term 'transitional infections'; two types will be discussed here, firstly, peach leaf curl and, secondly, loose smut of oats and leaf stripe disease of barley.

Taphrina deformans (peach leaf curl) attacks the young leaves, which become blistered and deformed. In the early summer it forms asci under the cuticle of the upper leaf surface and these produce and eject ascospores after which the fungus dies and the diseased leaves wither. On germination the ascospores give rise to a uninucleate budding mycelium which, since young susceptible leaf tissues are no longer available, maintains its existence at first only as a saprophyte on the scales of the next year's buds and on the bark of the branches, where it overwinters and so continues the source of infection into the following spring. Before the leaves unfold and even before they have developed a spongy tissue, these budding cells which are

frequently, but erroneously, termed 'conidia' reach the underside of the leaves. Here they drive a germ tube, and thus a hypha, into the middle lamella between the epidermal cells (Fig. 294), and then develop intercellularly through the young leaf tissue, and sometimes downwards through the petiole for a short distance into a branch (Mix, 1935).

At some time or other in the budding cells or more rarely in the tissue hyphae the binucleate phase is initiated by means of a simple nuclear division; in exceptional cases two uninucleate budding cells may fuse before infection and develop into a dikaryotic infection hypha. Later, conjugate nuclear divisions bring the number of nuclei in the hyphal cells in the leaf tissue to twelve or fourteen. Cell division occurs, however, before the development of the subcuticular ascogenous cells, each of which then contains a single pair of nuclei, which fuse in the ordinary way.

Thus, the life cycle of *T. deformans* includes a morphological change from a budding mycelium to a hyphal phase which is conditioned biologically rather than by the nuclei. In the budding mycelial phase the parasite lives from the summer until the following spring as a saprophyte on the branches and bud scales of its host although, doubtless, it could also grow on the branches of other trees. If the sprouting cells were not concealed among the bud scales, which renders chemical treatment difficult, the infection chain could easily be interrupted in this external saprophytic period by appropriate control measures. This saprophytic phase is the overwintering reservoir of infection for the attacks of the next spring and, if these succeed, the fungus occupies its host parasitically for about 3 months and produces its sexual fructifications.

Even though the fungus passes three-quarters of the year as a saprophyte on its host it is undoubtedly a true parasite because it can develop its perfect sexual state only during its parasitic phase. The long saprophytic phase is involuntary and forced upon it by the climatically conditioned development of the host. Because of seasonal changes there are, at the time of ascospore dispersal, no young leaves available for the continuous propagation of the infection chain; the fungus must, therefore, remain in abeyance as a facultative saprophyte until, in the seasonal rhythm of the host, susceptible organs once more develop. That this period can be spent on the surface of the host itself is a great advantage to the pathogen since it increases its chances of bringing about infection.

In the second group of examples, loose smut of oats (*Ustilago avenae*) (Kolk, 1930) and leaf stripe disease of barley (*Helminthosporium gramineum*), there are similar saprophytic transitional infections but, in these cases, they are strictly localized topographically.

The spores, brandspores in *U. avenae* and conidia in *H. gramineum*, are blown by the wind on to the viscid branches of the stigma where they germinate. The hyphae grow partly along the stigma lobes and partly within them down to the ovary, establishing themselves saprophytically mainly in the parenchyma of the glumes near the caryopsis but also in and on the epidermis of the caryopsis and the shrivelled remains of the pollen

sacs, &c. The incidence of infection is heaviest when the attack occurs immediately before flowering (Table VI).

TABLE VI

Relation between time of attack on barley by Helminthosporium gramineum and severity of infection. (After Genau, 1928)

Time of attack	Infection (%)	
	On Mahndorfer Hanna barley	On Heines Improved Chevalier barley
Shortly before flowering	46.6 ± 12.9	50.0 ± 11.2
During flowering	16.3 ± 5.6	34.9 ± 7.0
Immediately after flowering	16.3 ± 4.0	34.7 ± 4.8
Two days after flowering	9.1 ± 3.3	11.5 ± 3.1

Eventually the hyphae, especially in the apical region of the seed, develop into a thick-walled resting mycelium, which overwinters on the seed. It is very long-lived; for instance, Paxton (1922) observed the production in a moist chamber of germinable conidia of *H. gramineum* from 16-year-old herbarium material.

After sowing, the resting mycelium germinates with the seed (Fig. 90), attacks the young seedling either directly, or indirectly by contaminating the damp soil, and permeates it systemically. *U. avenae* gives rise to a smutting (see Fig. 98) and *H. gramineum* to yellow striping followed by brown discoloration and, later, shredding of the foliage leaves and a cessation of ear growth or sterility of the seeds. The infection chain of *Helminthosporium* can also start from ascospores produced in asci on the overwintered stubble which, in spring, attack the young foliage leaves directly from the soil. Because of crop rotation, however, this latter possibility is of little importance compared with the saprophytic transitional infection on the surface of the glumes and seeds.

Thus, in this second group of examples, two phases in the course of infection must be distinguished: one, the saprophytic transitional infection, leading through the formation of an overwintering saprophytic infection focus to a contamination of the surface of the seeds, and two, the actual infection of the young seedling itself. The transitional infection, like the budding mycelial phase in *Taphrina deformans*, merely provides the saprophytic starting-point on the surface of the host to be infected, brings the parasite into the immediate vicinity of the organs to be attacked, and maintains it until the time when the suitable tissues reach the correct stage of development.

The ultimate infection of the seedlings, however, is not really decided by this contamination, the chances of infection are merely enhanced. On account of the purely subsidiary nature of the transitional infection, the infection chain can be broken by control measures applied between the saprophytic and the parasitic phases. Thus, if the infected seeds are treated before sowing with mercury disinfectants or with hot air or warm

water, for example, for 5 minutes at 60° C. or 20 minutes at 55° C., the resting mycelium of the parasite is destroyed with no appreciable reduction in the germinability of the seeds, and the attack on the seedlings does not take place.

The actual infection of the host occurs, therefore, in the growing seedlings and not in the flower. However, through the production of a saprophytic focus of infection on the seed and glume surface, which originally came through the flower stigmas, the attack on the seedlings is decisively promoted. In practical plant pathology, therefore, this type of colonization is referred to as pseudo flower infection or sometimes as flower-seedling infection.

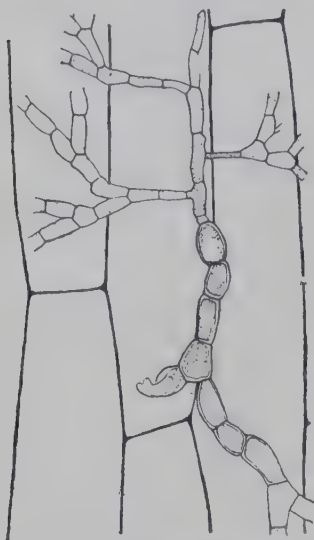


FIG. 90. Germinating thick-walled resting mycelium of *Helminthosporium gramineum* on pericarp epidermis of barley. $\times 350$. (After Vogt, 1923.)

(b) Saprophytic Phases in the Soil as Sources of Infection

Like the saprophytic phase on the surface of the future host discussed above, the saprophytic phase of the pathogen in the soil, compost, &c., is of an entirely facultative nature, i.e. it is not an essential developmental stage in the life history but a phase incidental to the parasitic mode of life. The parasite reaches the soil with the infected, decomposed remains of the host and for a time it may persist saprophytically. For example, three modes of subsistence are available to cereal pathogens such as *Ophiobolus graminis* (take-all and whiteheads, Fig. 91), *Cercospora herpocorticoides* (eyespot, Fig. 92), &c. After the harvest, i.e. following the death of the principal host, the parasites may (1) pass directly from root to root and propagate the infection chain continuously on auxiliary hosts, 'volunteer' cereals, and the wild grasses growing as weeds; or discontinuously (2), produce on the stubble in the course of the following year the ascus fructifications, the pseudothecia or (3), initiate a saprophytic phase and temporarily maintain life in the mycelial stage on the stubble until the new seed begins to germinate. In all three cases an enduring locus of infection for the succeeding crops develops in the infested fields.

Here we are concerned only with the third eventuality. Naturally the pathogens will only survive the saprophytic exile in the soil if they are as far as possible autotrophic in respect of growth substances, able to exist on a minimum of nutriment from their substrates, indifferent to external temperatures, and capable of overcoming the true saprophytic soil micro-organisms in the struggle for existence. The pathogenic bacteria of human medicine familiar to the layman are mostly too exacting in these respects, e.g. protein nutrition, optimum temperature about 37° C., &c. Hence, only the bacteria of the *coli*-group, which cause typhoid diseases, and the gas gangrene bacilli actually produce foci of development in the soil in the

botanical sense. Originating in faecal impurities, they can survive saprophytically in the soil, &c., for a limited period, the former for a few days or months, the latter at any rate for some years; they multiply and once more return to human beings. Most plant pathogenic fungi and bacteria, on the contrary, are less fastidious in this way and hence are better adapted to a more or less involuntary saprophytic phase in the soil.

On account of these frequent saprophytic reservoirs of development the soil forms an incomparably more durable source of contamination in the infectious diseases of plants than in those of man. Of saprophytic chthonic infections (p. 26), medicine has to fear only *coli* and gas gangrene, whereas the plant draws from the soil not only a proportion of its food but also a large number of its disease germs, being thus for good or ill more closely earth-bound than is man.

Where the same crop is grown in one field for several consecutive years, there is usually a progressive enrichment of the saprophytic infection reservoir in the soil and, therefore, in the choice of a rotation in agriculture and horticulture attention must often be directed in the first place to persistent sources of infection due to earlier diseases. Thus, in take-all soils the farmer does not sow wheat in immediate succession to maize or wheat, even if this be expedient on nutritional grounds, because wheat is usually highly susceptible to take-all; he interpolates the fairly resistant rye in the sequence.

He will revert to wheat only after an interval the length of which will depend (unless there are infection chains on subsidiary hosts to be considered) on the species of pathogen, the edaphic factors, and the other micro-organisms inhabiting the soil.

As regards the fungus species, adaptation and tendency to the saprophytic way of life vary greatly in degree among the several facultative plant parasites.

At one extreme are the pronounced saprophytes, living and proliferating freely in decomposed plant tissues and soil humus, where they produce all their spore forms, and only incidentally infecting certain specific hosts rendered particularly susceptible through debility. Thus, *Trametes radiciperda* (butt rot of spruce) builds up chronic saprophytic infection reservoirs in the stumps and humus of the diseased stands, where it also reproduces; it is, therefore, primarily a saprophyte but can pass to intact trees if these become specially susceptible. Similar conditions probably obtain in

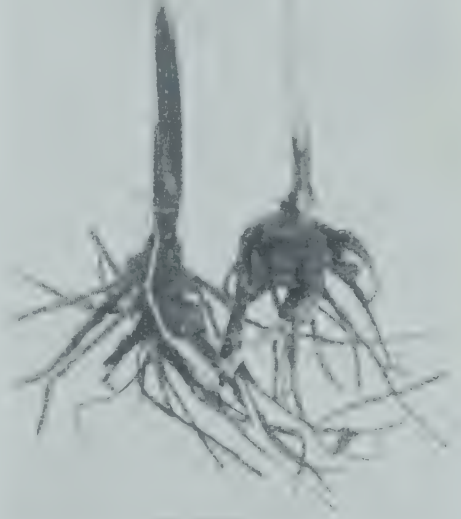


FIG. 91. Foot rot (take-all disease) of wheat caused by *Ophiobolus graminis*. Symptoms at the haulm base and on the leaf sheaths in mid-May. Approx. $\frac{1}{2}$. (After Oort, 1936.)

the case of such damping-off agents of many seedlings as *Pythium de Baryanum*, *Fusarium moniliforme*, *Corticium vagum*, &c. In forests these fungi are one of the principal causes of failure of natural regeneration; in agriculture and horticulture they are the cause of the epidemic collapse of sugar beet and vegetable seedlings, &c. Here, too, the parasites

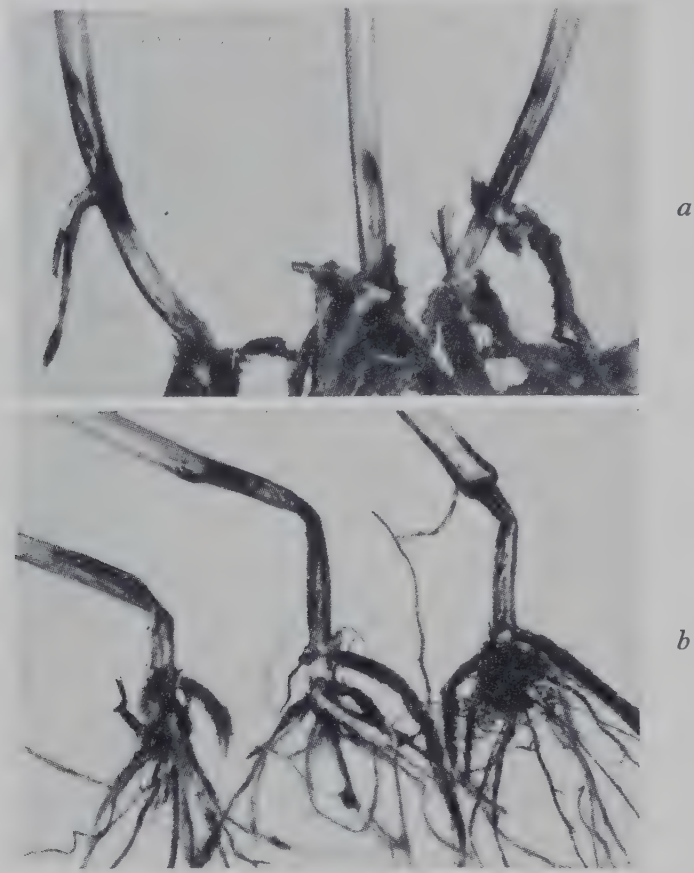


FIG. 92. The straw-breaking disease (eyespot) of wheat caused by *Cercospora herpotrichoides*. *a* Symptoms at the haulm base at the end of June; *b* lodged plants at the end of July. Approx. $\times \frac{1}{2}$. (After Oort, 1936.)

live primarily as saprophytes in the soil which, in consequence, is permanently contaminated by them and can only with difficulty be freed from their germs, or disinfected, by cultural means.

At the other extreme are the true parasites such as *Ophiobolus graminis*, which are ill adapted to a saprophytic life in the soil, in which they do not produce their higher, or sexual, fructifications, these only appearing on the dead or exhausted host organs. As will shortly be seen, the fungus in the soil dies out within a relatively brief period unless it can escape from its exile by again invading the host. The presence of the host ensures the continued existence of the pathogen which infects it.

Between these two extremes are innumerable disease agents in which the balance of saprophytism or parasitism is determined by the species.

As regards the relation of the second group to the soil factors, the maintenance of the saprophytic infection reservoirs is favoured or hindered by such conditions as the physical characters of the soil, its humus content,

reaction, and moisture relationships (Garrett, 1938, 1939), which the farmer seeks to improve, for instance, by soil cultivation. The degree of infectivity of the soil population is usually tested by the elective method, in which susceptible individuals are planted in the contaminated soil and the number of pathogens estimated from the relative amount and intensity of infection and disease. It is not always easy to separate the influence of the edaphic factors on the longevity and aggressiveness of the pathogen from their effect on the susceptibility of the host.

Physical characters of the soil. Among the cereal diseases, eyespot (Fig. 92), caused by *Cercospora herpotrichoides*, is most prevalent on heavy soils (Oort, 1936) and *Ophiobolus graminis*, the causative agent of take-all (Fig. 91), on light soils (Table VII). Thus, rolling leading to consolidation of the soil acts in an opposite way on two pathogens which produce very similar diseases.

TABLE VII

The growth-rate of Ophiobolus graminis along wheat roots in a sand-soil mixture. Duration of experiment about a fortnight. (After Garrett, 1936)

Parts by volume sand : soil .	Sand	1:16	1:8	1:4	1:2	1:1
Growth-rate of the fungus (mm.) .	53 ± 1.9	40 ± 2.3	16 ± 2.4	18 ± 1.8	6 ± 1.2	5 ± 1.4

Humus content of the soil. A certain minimum content of decomposable substances in the soil is essential to the saprophytic existence of any pathogen. An excess of organic matter due to addition of straw, compost, or green manure, inhibits such potato diseases as common scab (*Actinomyces scabies*) and black scurf (*Rhizoctonia solani*) probably through the competition of more vigorous saprophytes for food or the fungistatic action of their metabolic products. On the other hand, it benefits wheat bunt (*Tilletia tritici*) and the wilt disease of tomatoes, &c. (*Verticillium albo-atrum*).

Soil reaction. The results of laboratory tests on synthetic nutrient media cannot be applied directly to the soil because the hydrogen-ion concentration operates differently according to the nature of the substrate. Thus, *Pythium de Baryanum* (p. 98) and *Armillaria mellea* (p. 44) thrive best on acid soils, *Rhizoctonia solani* (Table VIII) on neutral soils, and *Ophiobolus graminis* (Table IX) on alkaline soils.

TABLE VIII

The influence of the soil reaction on the growth-rate of Corticium vagum (Rhizoctonia solani) in a loamy soil with a moisture content of about 35%. Duration of experiment about 5 days. (After Blair, 1943)

Soil reaction, pH .	3	4.5	5.8	6.3	7.1	7.4	8.1	8.7
Growth-rate of hyphae (mm.)	0	1.4	34	35	45	32	29	5

TABLE IX

The influence of the soil reaction on the growth-rate of Ophiobolus graminis along wheat roots. Duration of experiment about a fortnight. (After Garrett, 1936)

Soil reaction, pH .	4.8	4.9	6.8	7.2	7.7	8
Growth-rate of the fungus (mm.) .	8±1.1	11±1	13±0.6	20±0.9	25±1	28±1.3

Soil moisture. The soil-inhabiting pathogenic Phycomycetes which still form zoospores, e.g. *Pythium de Baryanum*, survive longer in damp soils whereas *Actinomyces scabies* (common scab of potato) is more persistent in those with a moderate water content.

The ambivalence of all these edaphic factors hinders the cleansing of infested soils by merely readjusting the environmental factors: there is always the risk of providing optimal conditions for a new pathogen, rather like casting out the devil with Beelzebub.

The third factor-group influencing the longevity of saprophytic infection foci in the soil comprises the soil micro-organisms, i.e. the edaphon. Three groups of partners can be differentiated on the basis of mutual relationships: competitors, satellites, and antagonists.

Simple, primitive competition is the most usual case. An intense rivalry for nutrients exists among various micro-organisms which, otherwise, neither favour nor hinder each other's progress by their metabolic products, to which they are indifferent. Thus, *Ophiobolus graminis* can exist saprophytically in the soil only until the stubble is decomposed and the nutrients exhausted; thereafter, it fails and disappears. Thus, it survives longest in the most unfavourable soils (Table VII), because in these the competitive micro-organisms are likewise strongly inhibited (Garrett, 1938). On the other hand, the measures that favour a prolific development of the true saprophytes and a rapid decomposition of the stubble, such as shallow cultivation of the field immediately after harvest, indirectly help to shorten the rotation interval.

Similarly, potato scab (*Actinomyces scabies*) is quickly reduced by mixing increasing doses of *Actinomyces praecox*, an obligate saprophyte, with the infested soil. The number of individuals of the parasite in the soil also falls immediately after the addition of the saprophyte (Millard and Taylor, 1927). Since the two micro-organisms are compatible in culture their incompatibility in the soil must depend entirely on competition for food; the more vigorous saprophyte starves out and supplants the parasite, unless the latter can withdraw into its sanctuary, the host.

In comparison with these more usual competitors for food only relatively few pairs of satellites are known. Their association generally depends on the donor organism supplying vital growth substances or hormones to its satellite, thereby enabling it to develop on soils poor in these substances (synergism).

For instance, *Ophiobolus graminis* requires a growth substance of the bios group, which is absent from the soil but present in the host organism. Thus, the refuge of *Ophiobolus* in its host does not present merely a negative aspect, the avoidance of competition with the saprophytes, but a positive aspect, the securing of the indispensable growth substance. *Ophiobolus* can, however, thrive in the soil provided that other micro-organisms, e.g. certain bacteria (Padwick, 1936), synthesize the particular growth substance. Among the parasites of forest trees, *Polyporus adustus* and *P. abietinus*, both of which produce white rots, the former on broad-leaved trees and the latter on conifers, are heterotrophic in respect of aneurin (vitamin B1) and must, therefore, obtain this from the substrate or from aneurin-autotrophic micro-organisms (Kögl and Fries, 1937).

This dependence of the satellites on their growth substance donors may entail the occurrence in the soil of a chronological sequence, a succession or metabiosis of micro-organisms because, on deficient soils, the satellite can only attain luxuriant development following upon that of the donor ('wet-nurse' growth).

In other cases the two satellite partners aid one another. Thus, both *Mucor Ramannianus* and *Rhodotorula rubra* need aneurin for their development. The former can synthesize only one fraction, pyrimidin, and must be provided with the other fraction, thiazol; conversely, the latter can synthesize thiazol but is heterotrophic in respect of pyrimidin. In joint culture the two fungi are mutually complementary and, therefore, are able to colonize substrates that would be unavailable to either of them singly (Utiger, 1942). Hence, since both partners derive benefit from their biological union, this is a true ('mutualistische') symbiosis.

The converse of mutual symbiosis occurs in the third group of micro-organisms, the antagonists, which inhibit or injure one another by their metabolic products (antibiosis). The growth-inhibiting substances are termed fungistatic or bacteriostatic and the lethal ones fungicidal or bactericidal. Some of these products, e.g. penicillin, secreted by the *Penicillium chrysogenum* and *P. notatum* groups, arrest the development of certain micro-organisms only; penicillin acts chiefly on gram-positive bacteria, especially cocci, but is harmless to man. These products are used in therapy (Wettstein, 1944; Hallauer, 1944).

In the laboratory testing of antagonism two partners are inoculated a certain distance apart on agar plates; where antagonism exists a 'free zone' in which neither of the micro-organisms grows readily is formed between the two colonies. Thus, the thermostable, acid-fast secretions of *Bacillus mesentericus*, a common soil bacterium, inhibit the growth of *Helminthosporium sativum*, the causative agent of a stripe disease of cereals, deform its hyphae, frequently induce sectoring even at low concentrations, and, whilst stimulating the production of conidia, hinder or prevent their germination (Christensen and Davis, 1940).

However, the results obtained on synthetic nutrient media cannot be applied directly to the environmental conditions of the soil. Thus, in culture, *Ophiobolus graminis* is completely inhibited by *Penicillium* F, whereas in the soil, notwithstanding the presence of the latter (Table X), it still attacks 30% of the wheat plants, perhaps because the antibiotic substances are decomposed by other micro-organisms. It is clear, therefore, that laboratory determinations of the degree of efficiency of the antagonism must be checked by practical infection experiments. In one experiment (Table X) sterile soil was contaminated with *Ophiobolus graminis* alone and also jointly with each of a number of saprophytic fungi and then sown with sterilized wheat. Whilst certain fungi, e.g. *Botrytis cinerea*, do not influence the development or pathogenicity of *Ophiobolus graminis*, others, such as a particular species of *Trichoderma*, are prohibitive.

TABLE X

The influence of various micro-organisms on the pathogenicity of Ophiobolus graminis. (After Lal, 1939)

<i>Pathogenic fungus</i>	<i>Additional, mostly saprophytic fungi</i>	<i>Infection of the primary roots</i>
		%
<i>Ophiobolus graminis</i>	—	100
	<i>Botrytis cinerea</i> Pers.	100
	<i>Acrostalagmus</i> sp.	90
	<i>Penicillium</i> D	81
	<i>Aspergillus</i> B	63
	<i>Penicillium</i> F	30
	<i>Rhizoctonia</i> sp.	24
	<i>Pythium</i> sp.	8
	<i>Trichoderma</i> sp.	0
	<i>Fusarium</i> A	0
Control	—	—

Similarly, the damping-off of citrus seedlings, caused by *Rhizoctonia solani*, can be arrested by a super-contamination of the soil with conidia of a soil saprophyte, *Trichoderma lignorum*, the young hyphae of which and of related *Trichoderma* spp. secrete an organic chloroform-soluble substance which is excessively toxic to *Rhizoctonia solani*, *Armillaria mellea*, &c. For *Rhizoctonia solani* the lethal threshold of this substance is at a dilution as low as 1:300,000, that is, it is about two-thirds as toxic as mercuric chloride (Weindling, 1934).

Many of the antagonisms are so pronounced as to suggest their application in horticulture for the biological control of plant pathogenic micro-organisms. Instead of applying the usual non-specific preparations of copper, sulphur, mercury, &c., it would be much more attractive to return to basic chemical principles, to isolate the specific toxins formed by the antagonists, find out their constitution, and then produce them synthetically in order to apply them as specific antidotes.

In view of their numerous antagonists and competitors the situation of

the plant pathogenic parasites during their saprophytic phase in the soil is not very favourable; the theatre of war is merely transferred from the host to the saprophytic infection reservoir. Most true parasites, however, are so poorly equipped for these new conditions that in the long run the host must serve as an asylum for them; thus both hosts and diseases are necessary to prevent the dying out of the pathogens.

3. *Resting States of the Pathogen as Sources of Infection*

The plant pathogenic viruses have no resting states and must, therefore, be propagated by continuous infection chains. True, the bacteria pathogenic to plants are also asporogenous but they can live saprophytically outside their hosts, for example, in the soil, whilst a few bacteria pathogenic to man, e.g. tetanus and anthrax, persist in the inanimate outer world by means of resting spores.

Of the pathogens which cause plant disease only the fungi possess resting spores or other similar resting states. Since the fungi are responsible for the majority of the infectious diseases of plants and since resting spores, &c., bridge the period (especially the winter) when the host is absent, these spores are of primary importance in plant disease in spite of their restriction to the fungi. Moreover, those disease organisms which under certain conditions can exist as saprophytic foci in the soil also generally have persistent resting states.

The morphological nature of these resting states differs according to the systematic position of the parasite; here we are concerned with resting spores, primordia of fructifications, and resting mycelium.

The period, generally winter, when a host plant is not available is passed in the form of resting spores by the great majority of Archimycetes (e.g. wart disease, club root), Oomycetes (e.g. oospores of downy mildew of vine, Fig. 93), Ustilagineae (brandspores), and Uredineae (teleutospores).

Plasmopara viticola (downy mildew of vine) may be used to illustrate the progress of an infection chain interrupted only by resting spores. In contrast to that of potato blight, this fungus overwinters in vine leaves on the ground by means of its thick-walled resting spores (oospores), which are surrounded by an oogonial wall, an exospore, and an endospore. They are found in large numbers (up to 200 per sq. mm.) in the leaf tissues during the autumn. They reach the soil with the falling leaves and, during the winter, are set free by the decay of the leaf tissues.

In the latter half of May or later, during heavy rain (about 10 mm.) when the temperature drops to 11° C., they germinate by means of a germ tube or a conidiophore. Within 3–6 hours this produces one or two large primary conidia (Fig. 93), which give rise to sixty or more zoospores. Rain-drops or spray carry these to the underside of the lowest leaves of the vine, where they push their germ tubes through the stomatal pores into the leaf tissues, so bringing about primary infection which is easily recognizable by the yellowish-green 'oil spots' on the leaves. A few days later the white conidiophores break through the under-surface of the leaves and, in turn,

abstract the smaller conidia. These are carried by wind or rain to fresh leaves, starting secondary infections, and thus introduce the continuous phase of the infection chain which lasts until the autumn when oospore formation again commences. Throughout this period diseased host plants constitute the only source of infection. The parasite spreads progressively farther in each generation and so brings about the summer epidemic of *Plasmopara*.



FIG. 93.

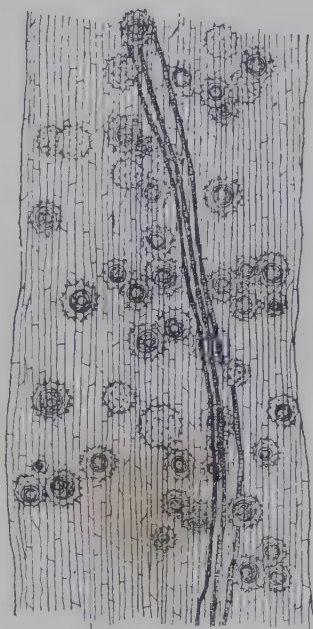


FIG. 94.

FIG. 93. A germinating oospore of *Plasmopara viticola* with primary conidium. Approx. $\times 500$. (After Arens, 1929.)

FIG. 94. Resting spores (oospores) of *Pythium megalacanthum*, the cause of a root rot of flax, in decaying roots. Approx. $\times 50$. (After Buisman, 1927.)

Morphologically, resting spores are generally recognizable by their thick, many-layered, cutinized and sculptured walls (Fig. 94), and also by their storage of fatty oils in oil drops. They occupy a definite position in the life cycle of the particular parasite, usually in direct or indirect relation to a sexual phase, and they are, therefore, generally diploid. More rarely, conidia overwinter, e.g. *Diplocarpon rosae* (black spot of rose), the perfect form of which is known only from North America.

Biologically, resting spores are resistant to cold; for example, the oospores of *Plasmopara viticola* can remain alive for 5 days at -20 to -26°C . They are resistant to drought and, in consequence, neither soil nor cereal can be disinfected merely by drying. Because of their cutinized outer wall they are also highly resistant to micro-organisms and survive the microbial activity in a compost heap, hence the spread of spores of maize smut, wart disease, club root, &c., from such heaps. Further, they are resistant to chemical action and are thus difficult to combat by chemical means. Their

germination is favoured by alternating cold and warmth, drought and moisture.

With the exception of the brandspores of certain Ustilagineae there is no obligate resting period and, under suitable conditions (which, however, are not realized in the open in our climate), the resting spores can germinate immediately they are formed, i.e. in the autumn. However, germination at the beginning of winter is generally difficult and slow and it proceeds more readily with increasing internal maturation (Fig. 95). Winter rest is, therefore, not an obligatory phase in the life cycle but a phase that is conditioned climatically.

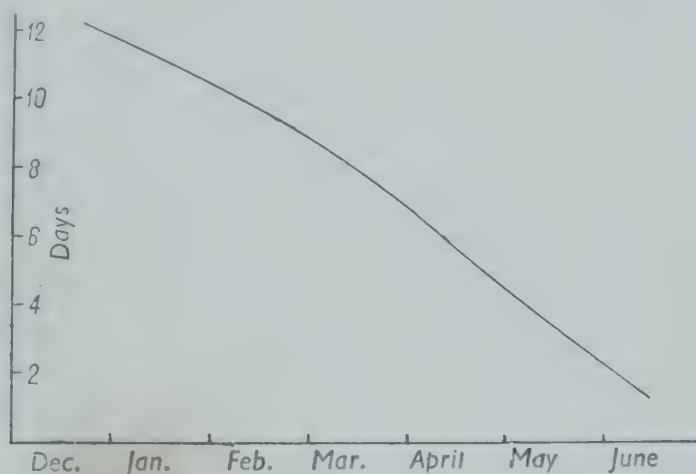


FIG. 95. The relationship between age of oospores of *Plasmopara viticola* and the time required for germination on damp filter paper; temperature 18–20° C. (Slightly diagrammatic, after Arens, 1929.)

The longevity of resting spores depends to a great extent upon external conditions but is sometimes astonishingly high. The resting sporangia of *Synchytrium endobioticum* can pass, unaltered, through the intestines of pigs and may then remain viable in the soil for about 10 years; hence the use of uncooked warted potatoes as feeding-stuffs enables the disease to spread via the dung. On the other hand, where weeds are not available to serve as interim hosts, the resting spores of *Plasmodiophora brassicae* (club root) remain viable in the soil for only 3 years. The brandspores of Ustilagineae, for example, can retain their vitality for a few years if stored completely dry in the laboratory, but under natural conditions generally lose it during the second year. The teleutospores of rust fungi are very sensitive to external conditions. They also can be stored in the laboratory for a long time, up to 6 years (Johnson, 1941), but lose their germinability in a few months if exposed to the influence of saprophytic micro-organisms; they are, therefore, ill adapted to withstand the rotting of the tissues of their hosts.

In nature, organisms causing plant disease can generally survive by means of their resting spores for only a limited period, seldom for more than 2 years, even under favourable circumstances.

The second kind of overwintering resting state, persistent fructification

primordia, is characteristic of some of the higher Ascomycetes, e.g. *Rhytisma acerinum* (tar spot of maple), *Lophodermium pinastri* (leaf cast of pine), and *Endostigme inaequalis* (apple scab). The rotting tissues of dead and fallen leaves contain a stromatic tissue, the fructification primordium, in which sexual processes occur, generally in autumn, leading to the formation of primary ascogenous hyphae. These overwinter and, in the following spring produce secondary ascogenous hyphae (Gäumann, 1940) which eventually bear ascospores that give rise to the primary infection. It is not the ascospores themselves that overwinter but the fructification primordia with their resting, primary, dikaryotic mycelium.

TABLE XI

The influence of cold (-1° C.) and of subsequent higher temperatures on the germination of ergots (Claviceps purpurea). (After Krebs, 1936)

Duration of exposure to cold (weeks)	Germination (%)			
	Germination temperature after cold storage ($^{\circ}$ C.)			
	9 $^{\circ}$	12 $^{\circ}$	15 $^{\circ}$	18 $^{\circ}$ and higher
2	100	98	0	0
3	100	98	0	0
4	96	92	0	0
6	82	78	0	0
8	66	62	26	0
10	34	86	46	0

Finally, the third method of overwintering, i.e. by a resting mycelium, can take place in one of two ways. In certain cases special morphological structures develop which, under certain circumstances, can be dispersed passively, e.g. ergot sclerotia (Fig. 81; Table XI), and certain sclerotia and bulbils of some of the Corticaceae (*Sclerotium Rolfsii*, &c.). In other cases, e.g. *Armillaria mellea* (honey agaric), brown hyphal strands or rhizomorphs are formed which spread from the stumps of attacked trees into the humus soil where they may persist for years and attack new trees whose roots become susceptible.

§ 3. The Transmission of Pathogens

How does infection travel? How are disease germs conveyed from distributors to receivers?

All infectious diseases are transmissible; otherwise they would die out with the diseased individual. Many are termed contagious which indicates either that they are very infectious or that they are very easily spread. The difference between infection and contagion may be illustrated by potato leaf roll. This is not contagious since the virus cannot be spread directly from one potato to another either by sap inoculation or by any kind of propagative unit; on the other hand, it is easily transmitted by aphides. Malaria in man forms a parallel case.

In the present chapter only the process of transmission is dealt with, the

last phase, the behaviour of the pathogen on the healthy plant, having been discussed under the process of infection in Chapter I.

The transference of pathogens from distributors to receivers takes place either directly, or indirectly through the interpolation of some means of transport; this section is divided, therefore, into two subsections: (1) direct, and (2) indirect transmission of pathogens.

1. *Direct Transmission of Pathogens*

According to the familial relationship existing between distributor and receiver and to the particular character of the transmission, direct transmission can be divided into:

- (a) germinative transmission; in which the young embryo is infected directly by the mother plant;
- (b) vegetative transmission; in which vegetative organs such as tubers and shoots are infected directly from the parent plants;
- (c) adherent transmission; in which the disease germs from the parent plant become attached to the surfaces of its reproductive bodies, fruits, seeds, &c., are conveyed about on them, and attack the young plants which emerge from them.

The germinative and vegetative types of transmission involve cyclic infections, i.e. infections which are transferred from parent to offspring within a closed circuit, the affected progeny being described ambiguously as suffering from a 'hereditary disease'. In contrast, the adherent and especially the indirectly transmitted germs give rise to spontaneous infections, i.e. infections that are entirely new.

(a) *Germinative Transmission of Pathogens*

In germinative transmission the offspring are directly infected by and within the parent plant so that the disease germ is spread by the seed (seed-borne transmission of disease). Ideally, this is found in cases where the future individual grows from an infected egg-cell. Thus, certain endobiotic bacteria and fungi, e.g. those living in the gastric appendages of cicadas and scale insects and often of value to their hosts as sources of vitamins and enzymes, are, in part, transferred to the offspring by the infection of the eggs in the ovary. In such cases, according to the principle of organotropic infection, the micro-organism returns by complicated ways to its pre-determined organ, e.g. a particular region of the gut (Buchner, 1940). Such possibilities may also arise in certain plant viroses and bacterioses but, as yet, their existence remains speculative.

Our knowledge is limited, therefore, to those kinds of disease in which the embryo is infected *in situ*; this process is analogous to intra-uterine infection in medicine.

In this group there are two possible modes of transmission: (*aa*) the diseased parent infects the young embryo, and (*bb*) the infected parent

transmits the disease germs to the young embryo without itself becoming diseased.

(aa) The diseased parent infects the young embryo. One example of a virus disease and one of a fungal disease will be given.

In addition to transmission by insects, bean mosaic can be spread by seed (Harrison, 1935). The virus, highly infectious because it can also be transferred by the pollen grains of infected plants to the stigmas of healthy

ones, evidently passes from the virus-diseased parents via the funicle into the seed primordium where it infects the young embryo. In this way there arises a germinative infection chain from parent to offspring, which is cyclic and inevitable.

Certain plants of darnel (*Lolium temulentum*) may become systemically infected by a fungus of the genus *Chaetomium* (Günnewig, 1933) without showing any obvious symptoms. The hyphae penetrate the seed primordia and later the young seedlings, so that these contain the parasite even prior to germination, i.e. there is cyclic infection.

(bb) The infected parent transmits the disease germs to the young embryo without itself becoming diseased. Economically, the most important examples of this are loose smut of wheat (*Ustilago tritici*) and loose smut of barley (*Ustilago nuda*). The brand spores are blown by the wind from

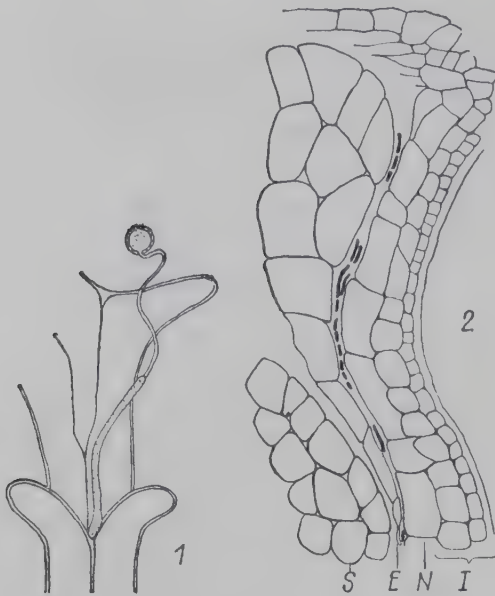


FIG. 96. *Ustilago tritici*. 1 A germ tube attempting to penetrate between the papillae of turgid stigmatic lobes of a wheat flower. 2 Seed primordium of summer wheat, 3 weeks after infection. S scutellum, E endosperm, N remains of nucellus, I integument, already strongly cutinized externally. Hyphae, indicated by black lines, occur between the endosperm and the seed coat (integument).
× 180. (After Lang, 1910.)

smutted ears to the stigmas of healthy plants, remain attached to the papillae, and germinate within 24 hours, producing a basidium; this at once grows out into hyphae which attempt to penetrate the papillae (Fig. 96, 1). So long as the papillae are fresh and turgid penetration is difficult, but when the stigmatic lobes begin to wilt (Fig. 97) and the cell tissues become flaccid, it is much easier for the hyphae to enter between the cells of the stylar canal and subsequently to reach the micropyle; this takes from 7 to 10 days. If the micropyle in the young fertilized seed be closed, the hyphae may grow through the inner integument (the outer having been resorbed shortly after fertilization) to the nucellus and between this and the young endosperm (Fig. 96, 2) into the scutellum, and thence into the young embryo.

They reach this about 4 weeks after infection and, during the following month, completely permeate the embryo and scutellum up to the vegetative

growing-point; the root primordia, however, remain free. Infection is now certain; up to this time the mycelium within the style or integuments might have been killed by a spell of warm, dry weather. Within the maturing grain the cells of the fungus swell considerably and their walls become thickened, i.e. the resting stage is attained.

On germination of the wheat or barley grains the parasite begins to grow intercellularly, immediately behind the growing-point into the culms, leaves, embryonic ears, and even into the roots. In the plants attacked it produces no obvious injury and correspondingly no special disease symptoms, but only a greater development of intercellular spaces, vascular bundles, stomata, and internodes, together with an increased transpiration and respiration rate. However, during the maturing of the host's flowers, spore formation takes place within the ears (see organotropic localization, p. 78) and results in the characteristic outbreak of the disease. The smutted ears protrude from the uppermost leaf-sheaths (Fig. 98), and the flowers, which are completely disorganized, are replaced by a dark brown mass of spores which, later, are freed by the rupture of the delicate silvery-grey membrane and, in their turn, are dispersed to new stigmatic surfaces.

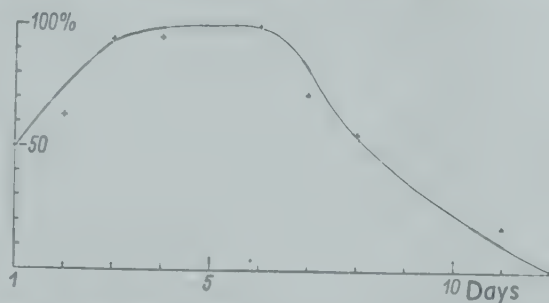


FIG. 97. The relationship between age of flower in Rümker's Sommerdickkopf wheat and its susceptibility to *Ustilago tritici*. Abscissae: number of days after the opening of the flowers, the first day is the actual day of opening. Ordinates: % incidence of disease. Diagrammatic. (After Piekenbrock, 1927.)

The primary entry point of the germs is normally the withered stigmas of the parent plant. If these stigmas do not become infected the infection chain is broken. If they do become infected this condition is purely transitional, i.e. the parent plant does not itself become diseased or show any symptoms but it merely transfers the pathogen through the style, &c., to its offspring. The parasite, therefore, passes down through the reproductive organs of the mother plant into the young embryo or daughter plant. In medicine such a course would be described as intra-uterine infection, a characteristically decisive infection of individuals which later become diseased. At this stage it is possible for the infection chain to be broken by a spell of warm weather or by hot-air treatment of seed stocks (p. 95) because, as in saprophytic germ transmission, the pathogen and host have different temperature requirements.

The infected offspring does not at once show symptoms of disease, but during its growing period it passes through a latent state of infection during which it becomes systemically permeated by the parasite. The actual disease appears in the flower primordia or the sexual organs of the offspring a full year after the primary infection of the mother plant.

The site of entry of the pathogen, the female reproductive organs of the parent plant, and the place where the disease breaks out a year later, in the

flowers of the offspring, do not correspond either in time or place. Furthermore, the tissues of the offspring, even in the embryonic state, contain hyphae from the infected though not obviously diseased parent. This paradoxical type of disease transmission is not known in human medicine.

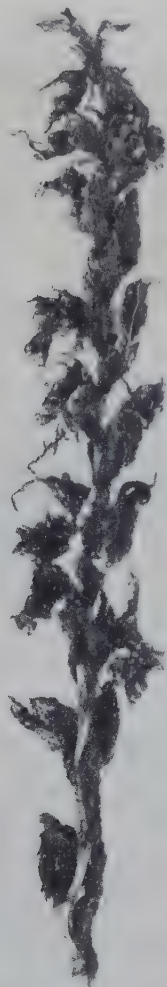


FIG. 98. Smutted ears of wheat, infected by *Ustilago tritici*. Nat. size. Orig.

(b) Vegetative Transmission of Pathogens

Vegetative transmission of disease is specifically a plant type of direct transmission since it is only among plants that new individuals are propagated vegetatively by tubers, cuttings, runners, grafts, &c. Systemic viroses and bacterioses, e.g. the disease caused by *Bacterium sepedonicum* (Fig. 62), are distributed throughout the entire parent plant and correspondingly infect its tubers, cuttings, &c., even before they become separated, so that a diseased clone results. Because of this cyclical infection of progeny, seed potatoes are sometimes harvested early in order that new virus infections in the leaves may be prevented from reaching the tubers. The infection chain in vegetative transmission can be broken only by the complete elimination of infected individuals from the plant and tuber stocks.

The bizarre conditions that may arise may be illustrated by reference to the King Edward potato. This variety of potato, one of the most frequently grown in Great Britain for the table, arose from a single seedling, which because of its valuable domestic qualities was propagated vegetatively from its tubers. The variety appeared to be free from virus as it never shows symptoms in the field and does not infect other potato varieties in neighbouring fields. Its cell sap, whether transmitted by insects or by wound infections, does not produce disease in other potato varieties. The symptom-free infection by the Z-virus, which nevertheless was present, would have remained undiscovered if experiments in another direction had not by chance revealed a complex interaction. Thus, if shoots of the King Edward variety are grafted on to plants of the variety Arran Victory, and if Y-virus be now superimposed, the stock becomes affected by a complex virosis, viz. paracrinkle (Fig. 155, left), characterized by dwarfing, large mosaics, and malformation of the leaflets (Salaman and Le Pelley, 1930). As every single King Edward plant is chronically infected with this symptom-free Z-virus it must already have been present in the original seedling, where possibly it arose spontaneously by mutation; owing to cyclic transmission all the derivative plants are infected.

This example of vegetative transmission illustrates the distinction that must be drawn between chronic and cyclic infections. Chronic infection

is a term used in individual medicine and indicates the persistence of an infection in a given individual. The expression cyclic infection has two meanings: as a concept of individual medicine it means a definite, regular spread of infection within an individual (p. 70), but as a term used in epidemiology it connotes a definite, obligate type of germ transmission.

In an epidemiological sense, cyclically infected individuals are generally also chronically infected, e.g. potatoes and their viruses. On the other hand, not all chronically infected plants transmit their disease cyclically. Thus, cypress spurge (Fig. 51) is chronically infected with *Uromyces pisi* but is not cyclically infective since it does not transmit the fungus to its progeny through the seeds, which are only rarely produced.

(c) *Adherent Transmission of Pathogens*

In the adherent type of transmission the disease germs pass from the parent plant to the surfaces of its reproductive bodies, the fruits, seeds, &c., which thus become contaminated and bear the pathogen along with them. Adherent transmission is, therefore, really a form of indirect transmission, because the reproductive bodies of the distributor serve as a passive means of transport for the disease germs. It occupies, however, a special place as in effect it comes closest to the germinative type, and is often confused with it. In both cases the disease germ is transferred to the immediate progeny, in germinative transmission as a true infection, in adherent transmission as a contaminant only. In order not to lose sight of this intimate connexion the adherent type of transmission is discussed under direct transmission.

Certain nitrogen-fixing bacteria, similar to *Bacterium radicola*, cause nodular tumours on the leaves of some tropical Rubiaceae, e.g. *Pavetta*, *Psychotria*, &c. The bacteria lying upon the leaf surface enter the mesophyll through stomata-like structures and initiate the development of cavities which, in part, may become filled in again by cell proliferation. The bacteria in question live as epiphytes on any part of the host plant, even upon its growing-point; they enter bud primordia and may become enclosed in the locules of the fruits, they can be found in the micropyles, and finally between the embryo and endosperm of the seeds. When the seed germinates the bacteria migrate on to the growing-point of the young plant and on to the leaves which they enter later (v. Faber, 1912). This is, therefore, a case of adherent transmission in which the parasite lives upon the host as an epiphyte prior to infecting it.

In one group of diseases of cultivated plants adherent transmission is responsible for the spread of the parasites by the seed stock. Examples of this are: wart disease of potato (*Synchytrium endobioticum*, Fig. 187), bunt of wheat (*Tilletia tritici*, p. 50), snow mould of cereals (*Fusarium nivale*), and, in a somewhat different sense, leaf stripe of barley (*Helminthosporium gramineum*, p. 94), loose smut of oats (*Ustilago avenae*, p. 94), and covered smut of barley (*Ustilago hordei*).

The seed is 'infected', more accurately contaminated, in so far as it bears the causal agent of the disease, though not within the embryo as in

germinative transmission. The brandspores of bunt become entangled in the beards of the grains, are dispersed with the grains, and finally return to the soil with them. The actual clinical infection which later gives rise to the disease does not appear, therefore, until the grain germinates and produces the seedling.

This adherent method of spread is controlled by keeping mycological watch on the seed stocks (Doyer, 1938; Neergaard, 1940) and by disinfecting them.

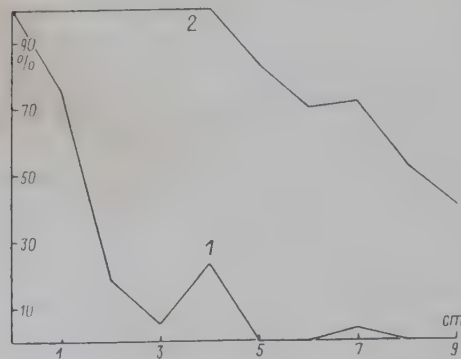


FIG. 99. The rate of spread of hyphae of *Rhizoctonia solani* (*Corticium vagum*) in clay soil with a water content of 43% and pH 6.8 infection. Abscissae: distance in cm. from site of infection. Ordinates: % mortality of radish seedlings. Curve 1: after 8 days. Curve 2: after 16 days. (After Blair, 1943.)

2. Indirect Transmission of Pathogens

In a few cases the pathogen actively and 'of its own accord' finds its way to the plants to be newly infected; these cases are grouped under (a) autonomous dispersal. The spread of plant disease is, however, normally accomplished by means of a definite transmitting agent or vehicle which passes from distributor to receiver. This transporting agency may belong to the inanimate world as in (b) anemochorous, and (c) hydrochorous dispersal, or to the animate

world as in (d) zoochorous and (e) anthropolchorous dispersal. In the two latter cases the transporting agent is termed a vector.

This section is divided into five subsections corresponding to these five primary modes of dispersal.

(a) Autonomous Dispersal of Pathogens

In entomology a noxious agent often spreads spontaneously over a wide area, but in phytopathology the active dispersal of a micro-organism is restricted to the immediate neighbourhood of the source of infection. Even where pathogens belonging to the plant kingdom possess a certain faculty of locomotion, e.g. flagellate bacteria, myxamoebae, and zoospores, the distances they can traverse of their own accord are infinitesimal by macroscopic measurement; their transmission more often takes place hydrochorously in water in which they support themselves by 'swimming'.

The autonomous dispersal of phytopathogenic organisms is accomplished, therefore, only by the active growth of hyphae and hyphal strands. Thus, under suitable conditions, the rhizomorphs of *Armillaria mellea* (honey fungus) may radiate from the stump of a dead conifer a dozen or so metres in all directions; in this way they can reach new hosts and, where local conditions are favourable, may not leave a single tree uninjured.

Much the same holds for the mycelium of *Corticium vagum* (Fig. 99), *Sclerotium Rolfsii*, *Phymatotrichum omnivorum*, &c. In *Herpotrichia nigra* (Fig. 71) the external infection hyphae can grow through the air.

(b) *Wind Dispersal of Pathogens (Anemochory)*

Wind is the most important agent of dispersal of plant pathogens, though only of fungi, not of bacteria and viruses. Many fungi produce on the outside of the host tissues, dispersal units capable of germination, the most important being conidia (Fig. 100), and these are spread by air currents, thus giving rise to aerogenous or air-borne infection (p. 26). Three problems arise here:

1. How do fungal spores enter air currents?
2. What are the physical conditions determining aerial transport of spores?
3. What is the range of successful anemochorous propagation?

1. How do fungal spores enter air currents? The fungus can play a passive or an active role in this event.

It occurs passively where the fungus merely abstracts its spores, or releases them in some other way, but has no mechanism for projecting them spontaneously. Thus, the dry powder of brandspores of loose smut of wheat (Fig. 98) is scattered merely by the shaking of the culms in the wind, or if the ears extend beyond the culms into the air, by direct wind suction.

In other fungi, e.g. the apple scab fungus (*Endostigme inaequalis*) and the agent of Dutch elm disease (*Ophiostoma ulmi*), the conidia (Fig. 73) are torn mechanically from their conidiophores by wind-driven rain or spray and carried with the drops in the wind for 10-20 m. To some extent, therefore, a droplet infection similar to that in medicine may be said to occur, but in man the mechanical conditions are quite different. In conversation, &c., drops of saliva containing suspended disease germs are sprayed into the surrounding air and re-inhaled by other people. Droplet infection in man, therefore, involves active participation both by the distributor and the receiver; in plants it is entirely dependent upon meteorological conditions. The microbiological relations are also somewhat different; in man it is predominantly bacteria and viruses that are transferred from distributor to receiver in droplet infection via the air, whereas in plants it is almost exclusively fungal spores.

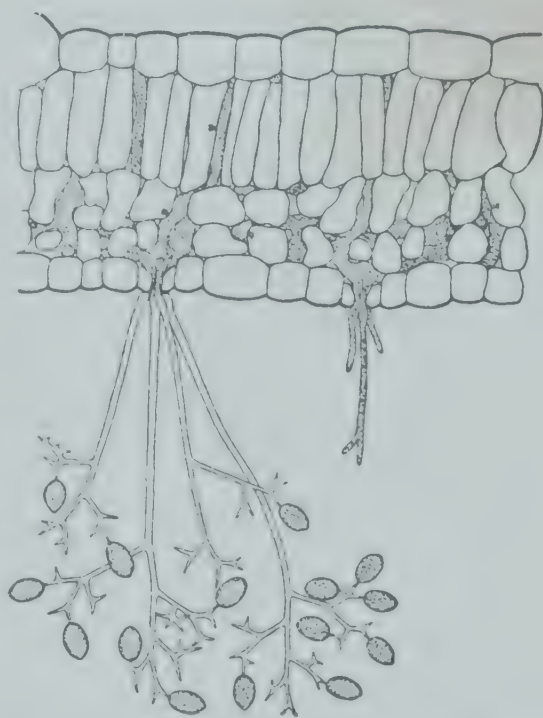


FIG. 100: Transverse section of a hop leaf infected by the mildew, *Pseudoperonospora humuli*, showing young and mature conidiophores and conidia. Approx. $\times 130$. (After Arens, 1930.)

In the majority of phytopathogenic fungi, which occur close to the ground surrounded by weeds and foliage, the chances of being carried upwards in wind eddies or spray are slight. The velocity of the wind close to the soil or to leaves, as near to any surface, is practically nil; hence, those types are favoured which actively propel their spores and so raise them out of the windless surface layers of air.



FIG. 101.

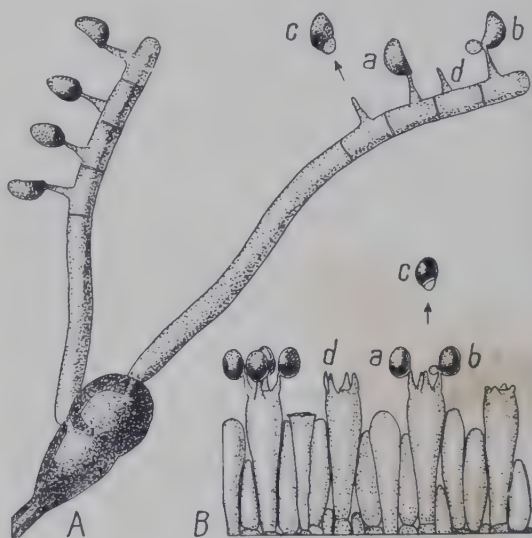


FIG. 102.

FIG. 101. The discharge of sickle-shaped conidia of *Tilletia tritici* (bunt of wheat). The brandspore *b* has germinated on the soil *a* forming a rudimentary basidium *c* which in its turn has produced 12 uninucleate basidiospores *d*. These have fused in pairs. The binucleate basidiospores thus formed, abstrict from the sterigmata *e* binucleate sickle-shaped conidia *f* and *g* which are discharged together with a drop of liquid *h* and give rise to fine binucleate hyphae which penetrate the delicate tissues of the seedlings. In a similar way sickle-shaped conidia are ejected from dikaryotic mycelium. $\times 670$. (After Buller and Vanterpool, 1933.)

FIG. 102. The successive discharge of basidiospores of *Puccinia graminis avenae* (black rust of oats, *A*) and *Psalliotia campestris* (mushroom, *B*). *a-d* stages in discharge. $\times 440$. (After Buller, 1924.)

Active discharge of spores has been established for numerous Phycmycetes, Ascomycetes, and Basidiomycetes. It involves a special mechanism both in exogenous spores abstricted from sterigmata, such as conidia, basidiospores, oidia, aecidiospores, &c., and in endogenous ascospores produced in asci.

A few seconds before the release of an exogenously abstricted spore a liquid droplet appears at its base, the delicate connexion between the sterigma and spore (hilum) is ruptured, and the spore together with the droplet is hurled for a distance up to a millimetre (Figs. 100-2). Aecidio-

spores, immediately after maturing, are also actively ejected from their cluster-cups so that in *Puccinia graminis*, for instance, it is often difficult to collect enough spores for infection tests.

In endogenously produced ascospores also, as in the case of conidia and basidiospores, the mother cells, here the mature asci, serve as an ejection apparatus.

At the beginning of the ejaculation period one ascus after another protrudes above the hymenium (Fig. 103, *a* and *b*), and the ascospores are shot out, in the operculate forms through a pore (Fig. 103, *c*), and in the inoperculate forms through the ruptured apex. The height of projection (associated ascospores usually clump together) may reach astonishing values, particularly when one considers that the fruiting bodies are, in general, scarcely 1 mm. high. Under favourable conditions of temperature and moisture a height of 15 cm. has been recorded for *Pleuraea fimiseda* and of 45 cm. for *P. curvicolla*; both of these are saprophytic dung fungi.

In other Ascomycetes, e.g. *Pleospora herbarum*, a plurivorous leaf and stem parasite and a saprophyte (Atanasoff, 1919), and in *Hysteroglyphium fraxini*, which is destructive to ash and olive trees (Zogg, 1943), expulsion is favoured by a differentiation of the ascus wall. This consists of two layers which first become visible at the moment of spore liberation; a stiff, inelastic, cutinized outer layer which does not swell in water, and a thicker, gelatinous inner layer which absorbs water and swells. The paraphyses also may swell. By the pressure of the inner swollen layer the outer one becomes torn at the apex and the inner one, together with the ascospores, bulges out, and with a sudden jerk ruptures laterally at the place of exit, or at the apex, and the contents are ejected.

In still other forms, e.g. *Endothia parasitica* (blight of chestnut and oak which occurs epidemically in the United States and to a limited extent in the Mediterranean region), and in certain Gnomoniaceae, the length of the perithecial neck does not allow the asci, as in *Pleuraea*, to reach the ostiole, and there to liberate their spores. They are released at the place of origin



FIG. 103. Ascospore ejection in *Sarcoscypha protracta*. The mature ascus *a* projects beyond the hymenium *b* ejects its contents and subsequently collapses *c*. *d* branched paraphysis, *e* ascospores. $\times 180$. (After Buller, 1934.)

and are squeezed through the perithecial neck, where the paraphyses doubtless play an active role. When the perithecial neck dries the asci become compressed, until they burst by lateral pressure and the spores are shot out. The mechanism of discharge is situated in the mouth of the perithecium and, if this be cut off, then emission of ascospores ceases, at any rate in *Endothia parasitica* (Heald and Walton, 1914).

The cloud of spores shown in Fig. 104, photographed with an exposure of one-hundredth of a second, gives an idea of the intensity of spore dispersal in *Sclerotinia sclerotiorum* (= *Scl. libertiana*), the cause of numerous 'Botrytis' diseases in storage organs, rhizomes,

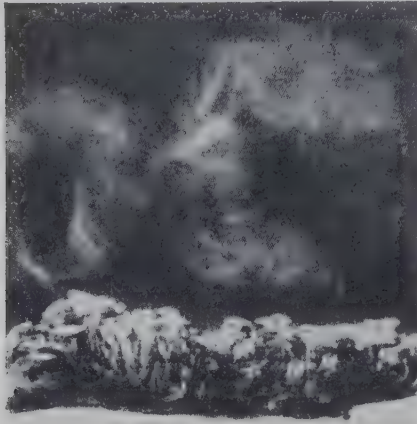


FIG. 104. A cloud of ascospores from discharging apothecia of *Sclerotinia sclerotiorum*. Approx. $\times \frac{2}{3}$. (After Dickson and Fisher, 1923.)

sugar beet, potato, &c. Similar conditions obtain in other pathogens as, for example, in *Endostigme inaequalis* (apple scab); thus, Frey and Keitt (1925) on a day in May when climatic conditions were suitable caught an average of $2\frac{1}{2}$ ascospores per litre of air. The tests were made at a height of 1 m. above the ground during a period of $8\frac{1}{2}$ hours in a wind velocity of 6–8 m. per second; the ascospores had been ejected from decaying leaves on the ground.

The period during which a fructification can emit ascospores varies; in *Endothia parasitica* it may continue for 168 days if moisture conditions are favourable (Heald and Walton, 1914). In practice, however, the time is much shorter, the main ascospore dispersal in *Endostigme inaequalis*, under central European conditions, being limited to the period from mid-May to the beginning of June.

2. The physical requirements for aerial transport of fungal spores. As indicated above, the problems set by aerial dispersal first arise at the moment when the spores have left the calm surface layer of air, i.e. when they have been discharged beyond this layer. The quantitative physical requirements for the aerial suspension of plankton (aeroplankton) have been studied by Schmidt (1925). In spore dispersal two opposite movements are involved: on the one hand there are the wind and convection currents set up by temperature changes, &c., which carry the spores, as well as dust particles, upwards, and on the other hand the falling or downward movement which, on the whole, follows Stokes' law. The time and distance of spore dispersal are inversely proportional to the square of the velocity of fall in an undisturbed atmosphere.

The weight of a fungal spore is in the region of hundredths of a microgramme. The rate of fall of a basidiospore of *Lycoperdon Bovista* (giant puff-ball) in a still atmosphere is about 0.05 cm. per second (Schmidt,

1925), and that of the uredospores of the heavier cereal rusts *Puccinia graminis*, *P. coronata*, and *P. triticea* is about 1 cm. per second (Ukkelberg, 1933). In the ordinary course of events on the earth's surface, the average range of flight of the very small basidiospores of *Lycoperdon Bovista* is about 460,000 km. (Schmidt, 1925), i.e. a good eleven times the circumference of the earth, and that of the uredospores of cereal rust about 1,100 km. There are, therefore, no physical limits to the dissemination of spores in the atmosphere; the slightest breath of wind, physically almost too slight to measure, is sufficient to whirl them about afresh.

It is not easy in any single case to determine whether the range of flight of a fungal spore is greater at low or at high wind velocities. At low velocities air pockets of turbulent, unco-ordinated movements arise, so that spores may be whirled to the upper strata of the air (Fig. 105, *B*) and may, perhaps, be scattered over continents. At high wind velocities the air movements are chiefly laminar and co-ordinated, so that spores are more likely to remain at soil or plant levels (Fig. 105, *A*) and come to rest a short distance away; as against this, high velocities counteract more strongly the tendency to fall.

3. The practical range of anemochorous propagation. In infectious diseases of man, droplet infection in the open, as mentioned on page 113, is effective only when the distance between distributor and receiver is short; it is restricted, therefore, chiefly to short-range infection. On the other hand, in many fungal diseases of plants dissemination over wide areas is possible, i.e. there is long-range infection.

This problem of range comprises two subsidiary questions which are often confused, although they lead to opposite conclusions: (*aa*) how great is the effective economic range of a single source of infection and (*bb*) epidemiologically, how far can the first link of an infection chain extend?

(*aa*) The effective economic range of a single source of infection. It is clear that the density of spore dispersion lessens considerably with the distance between distributing and receiving agents; even at a distance of only 100 m. from the dispersal centre an enormous number of spores is required to contact one leaf with a reasonable degree of probability. The largest possible quantity of inoculum reaches the receiver in short-range infection and this is, therefore, much more effective in plant pathology

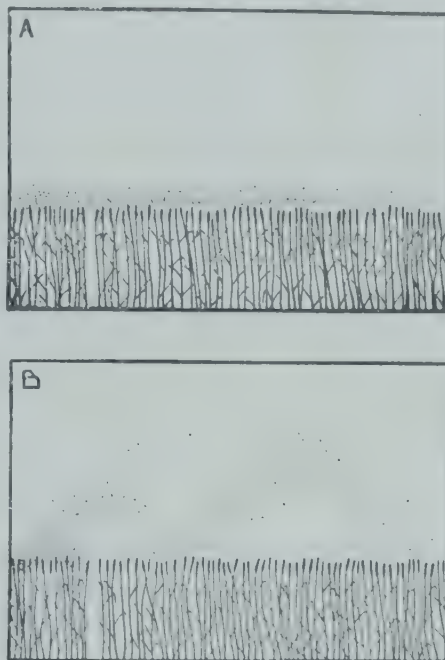


FIG. 105. Diagrammatic representation of anemochorous dispersal of the basidiospores of *Ustilago tritici* (loose smut of wheat), over a wheat field. Wind movement from left to right. *A* strong wind, *B* slight wind. (After Oort, 1940.)

than long-range infection. Other things being equal, a plant disease occurs in greatest amount and severity around its original source of infection.

Fig. 106 is a graphic representation of the decrease in infection density corresponding to increasing distance from the source in *Cronartium ribicola* (blister rust of Weymouth pine), on the basis of field trials over several years in a closed pine plantation in Idaho. Heavily infected bushes of *Ribes lacustre* and *R. viscosissimum*, on whose leaves the teleutospores overwinter, served as the source of infection. In the late summer or autumn the germ tubes of the basidiospores infected the needles of *Pinus monticola*

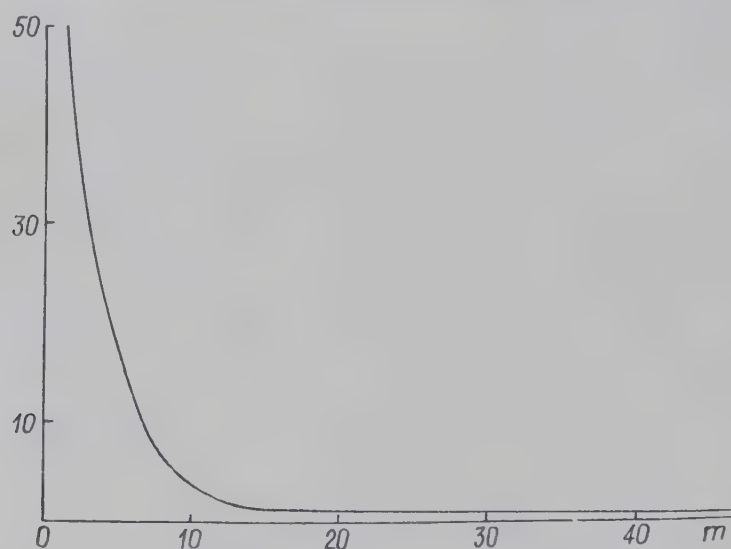


FIG. 106. The individual economic dispersal range from an infection centre (short-range infection) in *Cronartium ribicola* (blister rust of Weymouth pine). Explanation in text. (After Buchanan and Kimmey, 1938.)

(western white pine) which, like the Weymouth pine, is highly susceptible to blister rust, and then grew down into the bark of twigs and branches (p. 52).

The abscissae show the distance of the pines from the centre of the *Ribes* bushes; the ordinates give the number of pustules per million pine needles, i.e. the number of successful infections. At a distance of 1.5 m. there were 46 pustules per million needles, at a distance of 10 m. there were 5 pustules, and at 15 m. only 1; from this point onwards the incidence of disease remained more or less constant for a radius of about 50 m. Although, therefore, blister rust forms a vast number of teleutospores on *Ribes* leaves and although these spores germinate to give four times as many basidiospores, yet the dispersal density and thus the probability of contact fall steeply to a low limiting value even at a distance of only 15 m. from the source of infection.

However, should certain winds prevail, as in valleys or coastal lowlands, then spore dispersal will be influenced and may extend considerably farther on a narrower front. Thus, in Holland, a quadrangular infection centre of loose smut was established in the middle of a large wheat field. The wind carried the smut spores to healthy plants which were harvested; in order to

determine the sequence of infection these plants were sown in groups the following spring. Fig. 107 indicates that in the area in question, under a prevailing north-west wind, a serious infection band of over 100 m. long had to be taken into account.

These figures, 15 m., 100 m., &c., give the range over which a single centre of infection can produce direct economic injury and loss. The second problem is quite different.

(bb) The epidemiological range of a source of infection. How far can the wind transmit an infection chain from such a source?

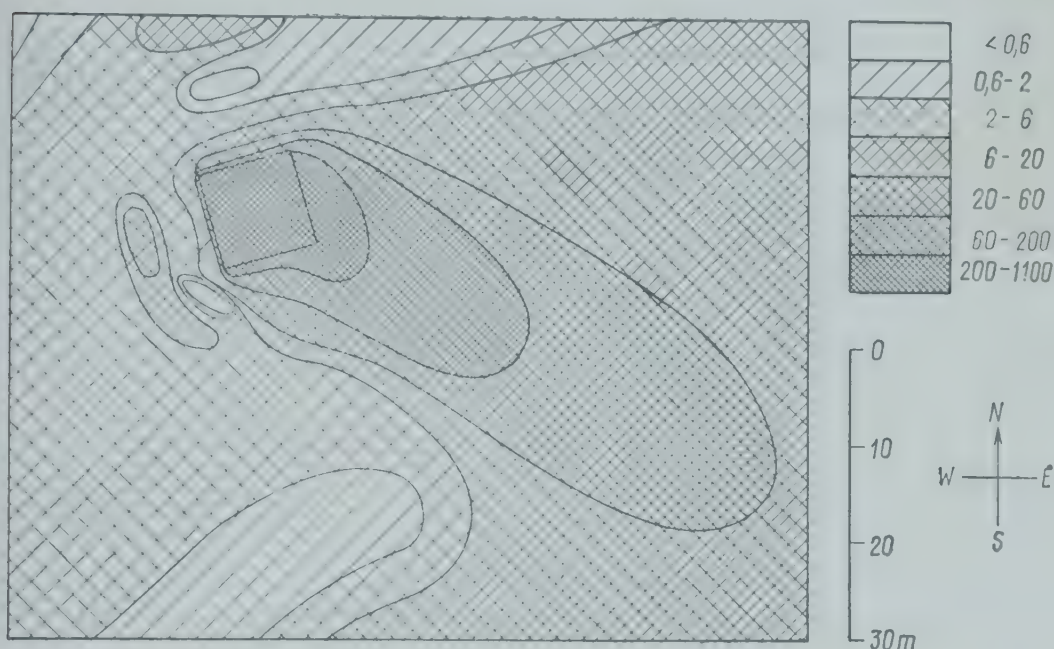


FIG. 107. Anemochorous infection of a field of wheat by *Ustilago tritici* (loose smut), under a prevailing north-west wind. Quadrat: focus of infection. Isobars: number of infected ears per 100 sq. m. Explanation in text. (After Oort, 1940.)

This is one of the key problems of progressive epidemics. For example, neither the break in the steeply descending curve of Fig. 106 nor the inner dispersion ring in which the density is still sufficiently high to cause immediate loss to the landowner is significant. The decisive point is the termination of the level prolongation of the right-angled curve in Fig. 108, which corresponds to the limiting value of spore dispersal. It is not the millions of spores lost by the way that are important but the few which reach their goal at the periphery of the dispersion zone and are still capable of infection. For the owner of the plants concerned the infections they produce here may be inconsiderable but, epidemiologically, these sparse and long-range infections are important because they extend the infection chain by leaps and bounds.

Since, in progressive epidemics, it is the maximal range of germinable spores that is decisive, the numerical values of epidemic significance relating to wind dispersal of spores are of quite a different order from those in Figs. 106 and 107. For example, in blister rust of Weymouth pine (Fig. 108), the maximum dispersal range inside the plantation is at least 500 m.

In the open field it is naturally much greater. In the summer of 1922 in a district in Minnesota there were 175 *Berberis* bushes in seven groups (Fig. 109). The distribution diagram shows that the aecidiospores of the black rust spread effectively over an area with a radius of at least 3 km. from the infected barberry bushes. If, therefore, the control of an epidemic be attempted by eliminating the alternate host, this extent of possible effective spread of spores must be taken into account.

Even greater epidemic ranges of aerial spore dispersion exist where the germ reservoir extends over a whole continent, e.g. the brown and black

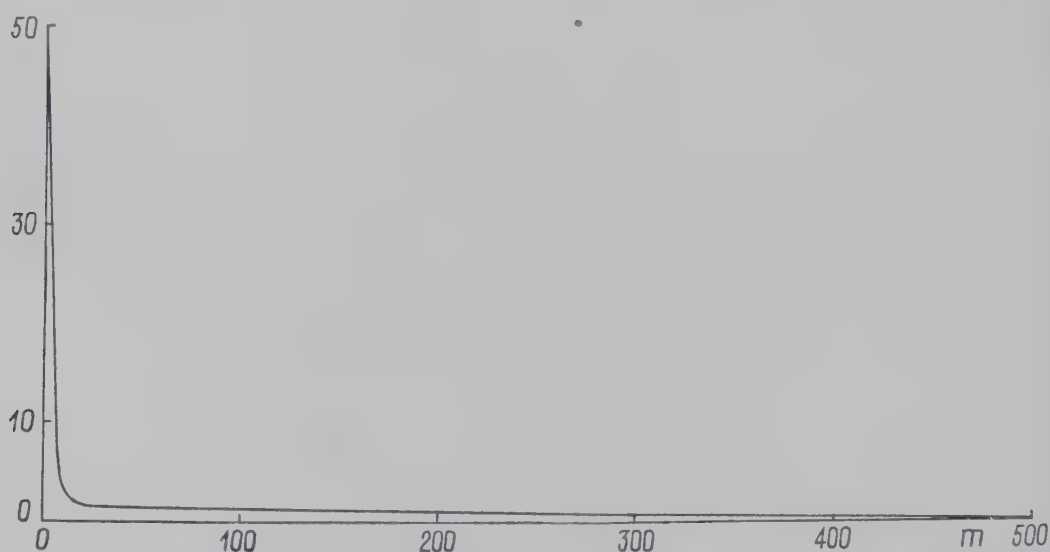


FIG. 108. The epidemiological dispersal range (long-range infection) in blister rust of Weymouth pine (*Cronartium ribicola*) in the interior of a wood. Explanation in text. Diagrammatic.

rusts in the North American wheat areas. In this case the entire atmosphere is contaminated with an aerial plankton of pollen grains and the spores of these pathogens. For instance, above the Mississippi valley aeroplanes caught uredospores of *Puccinia triticina* (brown rust of wheat) at 4,950 m., spores of *Alternaria* spp. at 3,150 m., and aecidiospores and uredospores of *Puccinia graminis* (black rust) at 2,100 m.; all these spores were still viable (Stakman *et al.*, 1923). Hence these rusts can overwinter in the southern states and be transported in spring in the upper layers of the atmosphere to the northern wheat areas.

(c) Water Dispersal of Pathogens (Hydrochory)

True water dispersal, i.e. the spread of disease germs by flowing water and not by rain splashes (see p. 113), is rare in plant disease although it may be expected to occur with soil-inhabiting species of *Pythium*, *Fusarium*, plant pathogenic bacteria, &c., in small areas after heavy summer showers.

The ergot sclerotia of aquatic and swamp grasses (*Glyceria*, *Molinia*, *Phalaris*, *Phragmites*) can float because of their air content and are thus dispersed by water (Stäger, 1922). On the other hand, the ergots of the forms of *Claviceps* on land grasses cannot float and immediately sink to the bottom.

(d) Animal Dispersal of Pathogens (Zoochory)

Animals are specific vectors if they transmit only one kind of disease germ and non-specific if they transmit several; for example, the peach aphid transmits several potato viruses.

If the vector merely transports the pathogen and, perhaps, also deposits it in the new host, then it is a carrier, a term which is the homonym of symptomless carrier (see p. 90). If, on the other hand, the pathogen under-



FIG. 109. The intensity of attack by black rust of wheat in the neighbourhood of seven groups of barberry bushes. Explanation in text. (After Stakman *et al.*, 1927.)

goes development inside the vector's body, then the vector serves as an alternate host. Thus, in relation to potatoes, the peach aphid (*Myzodes persicae*) is a carrier of the Y-virus (causing necrotic streaks along the leaf veins, 'raspberry leaf', stunted growth, and a shrivelling of the leaves on the plant), of the A-virus (causing a light paling of the veins which gives rise to a slight mosaic), and of the F-virus (causing yellow spots or aucuba mosaic, and net necrosis in the tuber), whereas it is an alternate host for the leaf roll virus (p. 87).

However, a sharp distinction cannot always be drawn between carrier and alternate host. Throughout their lives many carriers harbour and support certain disease germs and form an essential reservoir for them and thus, according to the wording of the definition, they might be termed either carriers or alternate hosts. The botanist generally understands the term

alternate host in the narrow sense of those individuals in which the pathogen undergoes a definite and specific phase of development; all other hosts are accordingly termed carriers or subsidiary hosts.

The present section deals exclusively with animals as carriers of disease germs and not as alternate hosts; the latter have already been considered on page 87 in connexion with heterogeneous infection cycles or change of host. The role of insects as vectors has been admirably described by Leach (1940).

According to the site, e.g. the part of the body which the disease germs occupy on the vector, the transference of germs by animals is effected epizootically (epizootochory) or endozootically (endozootochory). In epizootic transmission the disease germs adhere, like dust, anywhere on the surface of the vector which transports them directly to the receiver. In endozootic transmission, on the other hand, the vector harbours the disease germs inside its body and may nourish them from time to time; the association between the vector and the pathogen is very much closer. It follows that epizootic vectors are mostly non-specific whereas endozootic vectors are often specific for particular germs.

According to the method or the mechanism by which the vector delivers the disease germ to the receiver a distinction can be drawn between contact transmission, in which the vector is merely an agency of transport, does not harm the receiver, but merely deposits the disease germ superficially, and wound transference, in which the vector also wounds the receiver and implants the disease germ in the wound. Wound transference vectors are themselves already injurious to plants but their own damaging effect is incidentally increased because, at the same time, they also infect the injured plant with disease germs.

Although these local and mechanical possibilities of disease transmission are not, in reality, always so easily distinguished as the wording of the definition would suggest, their combinations give four fixed points around which characteristic instances can be grouped. The present section is, therefore, divided into (1) epizootic contact transmission, (2) endozootic contact transmission, (3) epizootic wound transmission, and (4) endozootic wound transmission.

1. Epizootic contact transmission of disease germs. The epizootic contact transmission of phytopathogenic germs is usually connected with the feeding habits of the vectors. The best-known example of this kind of incidental dispersal is the mistletoe (*Viscum album*). The mistle-thrush (*Turdus viscivorus*) and other migratory birds return from North Africa during the late winter or early spring and on the way they eat, among other things, mistletoe berries which have ripened during the winter. Many of the viscous, slimy seeds stick to their legs or feathers during meals, others are swallowed and pass uninjured through the alimentary canal; when their bearer alights on a new tree they are deposited on branches where they germinate (Fig. 10). As mistle-thrushes can fly over 100 km. in a few hours, they spread the seeds over wide tracts of land during their migration after

the winter. Their importance for the transport of mistletoe seeds also accounts for the extraordinary coincidence between their migratory routes and the geographical distribution of mistletoe, e.g. in south and west Switzerland (Tubeuf, 1923).

In the case of ergot of grasses (*Claviceps purpurea*) the host plant or distributor secretes as a honeydew (Fig. 82) a juice containing sugar which it can no longer use in the formation of endosperm, this having been destroyed by the parasite. The juice attracts all kinds of non-specific vectors such as flies, midges, &c., which, when feeding, pick up the conidia of the fungus which are embedded in the honeydew and deposit them on new stigmas. This transmission can occur either externally or internally; in the latter case the conidia are taken into the alimentary canal and later passed out in a still viable condition. Similarly, in North America the pollinating bees and flies that visit the nectaries of apple trees, &c., convey along with the pollen grains the causal agent of the dreaded fire blight (*Bacillus amylovorus*, Fig. 15).

In other cases it is simply attractive odours that cause the insect to visit the source of infection; they become contaminated superficially and thereby spread the pathogenic agent. For instance, the oidial pustules of the *Sclerotinia* spp. (p. 82) living in the Ericaceae smell like almond blossom. A similar sweet scent arises from the pycnidia of many rust fungi; this attracts insects and facilitates diploidization, and consequently the further development of the rust.

In still other cases there is neither attraction nor any kind of mutual benefit between the parasite and the host or distributor. The transport is provided quite unintentionally by the vector which becomes accidentally contaminated with spores, &c. Heald and Studhalter (1914) obtained spores of *Endothia parasitica* (p. 115) from 19 out of 36 species of bird that were found on or near diseased *Castanea* trees; in one case more than 700,000 spores were present. The cultures which were made from single birds showed that there were 2-14 times as many viable *Endothia* conidia as of all the other kinds of fungi together. Birds are also regarded as a dispersal agent of the perithecia of some Erysiphaceae, e.g. *Sphaerotheca mors-uvae* (American gooseberry mildew).

2. Endozoic contact transmission of disease germs. Endozoic contact transmission seldom occurs in plant pathology because the vectors, mostly insects, that are infected internally do not merely deposit the disease germ in their faeces or vomit on an undamaged receiver; they injure, bite, or colonize the latter at the same time. Hence, these vectors must be grouped with the endozoic wound transmitters.

An unusual instance of endozoic contact transmission is shown by the fungus genus *Septobasidium*, in which the stroma (Fig. 110) develops little if at all unless it be associated with certain scale insects, when it develops readily.

Septobasidium Burtii lives in association with the scale insect, *Aspidiotus osborni*, on oaks in North America. Allied species occur in Switzerland on

other deciduous trees on the bark of which they form stromata several centimetres in diameter. These stromata are attached loosely and are easily removed except where insects are present. The latter are covered on the outside by the fungal network (Fig. 111) whereas, on the underside, their sucking mouth parts penetrate into the bark; in consequence the fungus is attached to the substrate by the sucking organs of the insect. The fungus is primarily a parasite of the scale insect and only secondarily of the host plant; in any case the latter relation is half epiphytic. When they are present in quantity these fungal stromata can be very troublesome in tropical plantations of *Thea* and *Cinchona*.



FIG. 110. Encrustation of *Septobasidium bogoriense* on *Erythrina microcarpa*. (After Gäumann, 1922.)

Infection of young scale insects takes place, for example, at the base of the setae and the hyphae and budded cells are carried away in the blood stream or haemocoel and develop in a complex way during the different ecdyses (Couch, 1938). Finally, they again emerge into the open and on the bark form a thick, fungal stroma which is characteristic for each species and which later bears reproductive bodies. The insects parasitized by the fungus are retarded in their development, usually do not form scales, and are seldom able to reproduce, but on the other hand hundreds of uninfected scale insects overwinter in the cavities and spaces of the fungal web.

The fungus as well as the colony of scale insects gains certain advantages from this slave-like association. The fungus absorbs food from the parasitized insect which has sucked it out of the host plant and further elaborated it. The colony of scale insects, even though individuals are sacrificed, is provided with a home for the countless non-parasitized individuals which are protected from heat, drought, birds, and ichneumon flies. In considering why the fungus should build such a peculiar home for the insects one must remember the close relationship between scale insects and gall-inducing insects. The habitations are only built in the presence of living insects which, themselves, may supply the stimulus.

3. Epizootic wound transmission of disease germs. The wasps provide an example of this group. During the summer their biting mouth parts become contaminated from *Monilia*-infected places on ripening stone

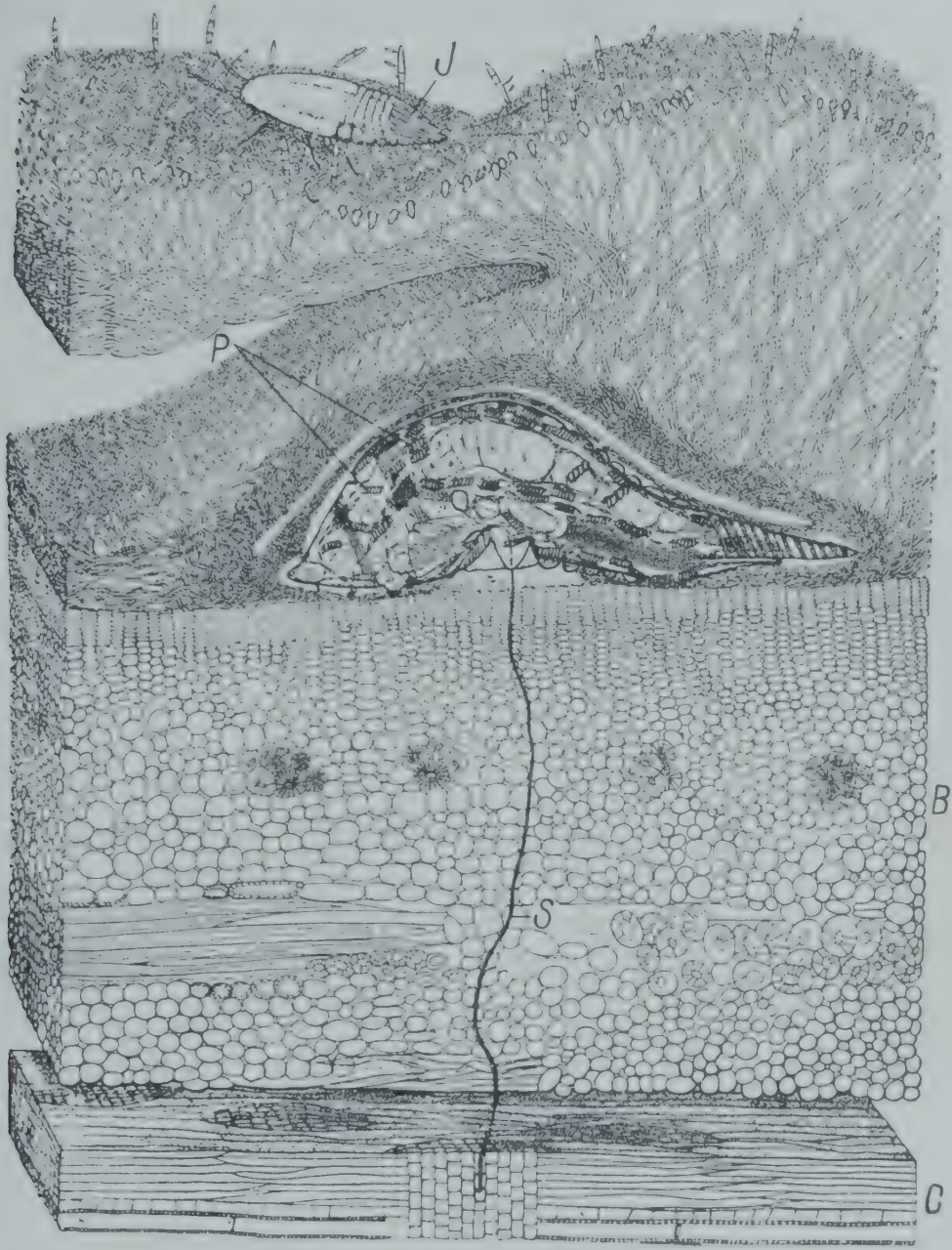


FIG. 111. Section through a colony of *Septobasidium Burtii* on the bark of an oak. The sucking mouth part *S* penetrates through the cortex *B* into the cambium *C*. The fungus *P* produces many coiled hyphae inside the host insect and on the outside of the bark interweaves to form a thick crust on which the young scale *J* crawls around and becomes infected by basidiospores. Semi-diagrammatic. $\times 100$. (After Couch, 1938.)

fruits. Subsequently, the wasps fly to undamaged fruit which they bite and the *Monilia* conidia are thus deposited in the wound, a favourable site for germination (Fig. 112).

Another example is the coral spot disease of spruce caused by *Nectria cucurbitula* which assumes epidemic proportions after a heavy attack by the pine bark moth.

In both cases the vector conveys the disease germ on the surface of its body and inserts it in the wound caused by its bite.

4. Endozoic wound transmission of disease germs. This type of germ transmission by animals is of special interest biologically because it involves an intimate interplay or even a vital association between a particular parasite and a specific vector. The latter not only conveys and introduces the disease germs, as in epizoic wound transmission, but it is at the same time a constant reservoir and, to a certain extent, a subsidiary host. The infection chain can, therefore, be interrupted only by successful control of the vector which forms the reservoir.



FIG. 112. Epizoic wound transmission of *Sclerotinia fructigena* to a ripe pear. The infection originated at the black spot. The fungus has formed its grey pustules of *Monilia* conidia in concentric rings. Approx. $\times \frac{2}{3}$. Original at the Federal Research Institute, Wädenswil.

In contrast to *Septobasidium Burtii*, the parasites concerned here are not pathogenic to their vectors; the latter do not themselves suffer, but in many cases derive positive advantages. The germs which are transmitted are of vital importance for the nutrition or growth of the vectors and are, therefore, preserved, cultivated, or otherwise favoured by them although they are also pathogenic to the host plants of their vectors, a fact of economic importance. In this sense, the small section of endozoic wound transmitters forms an exceptional case in the broad group of insects and the micro-organisms that inhabit them (Buchner, 1930). In only a few cases do the endozoically inoculated disease germs seem to be entirely without significance for their vectors.

Thus, the ambrosia fungi serve for the nourishment of their vectors; they are cultivated in the burrowing holes and feeding passages of certain bark beetles and are obviously given to their brood as food rich in vitamins or as 'fodder yeast'. Some of them, however, are pathogenic for the host plant of their vectors. Thus *Ophiostoma ulmi* is cultivated by the elm bark beetles (*Scolytus scolytus* and *S. multistriatus*) in their brood chambers (Fig. 113) as ambrosia fungi (Fransen, 1939) but, incidentally, this fungus also causes the death of elm trees owing to its toxic metabolic products. Other species of *Ophiostoma* (syn. *Ceratostomella* or *Graphium*) are reared by the bark beetles of conifers and cause a blue rot of the standing and felled tree trunks.

Neither group of fungi is necessarily dependent on vectors for its transmission and furtherance since the spores may be scattered by rain or wind and may infect through wounds or frost injury; the most effective dissemination is, however, by means of the vectors mentioned above.

On the other hand, the vectors seem to be entirely dependent on these fungi, perhaps for the supply of growth substances, because they have not yet been reared successfully under sterile conditions, i.e. in the absence of the fungi. Nevertheless, the association between the vector and the micro-organism is not a particularly close one. The young beetles are not infected with ambrosia fungi directly from the mother's body but first receive them from outside in the brood chamber. They then carry the



FIG. 113. Larvae and feeding passages of the elm bark beetle.
Approx. nat. size. Original.

spores of the fungi under their skeleton (Fig. 114) or in their alimentary canal (Fig. 115) to the new brood chambers where the spores in a fully germinable condition are voided in the excreta.

A similar physiological-nutritional relationship is provided by the canker-producing bacteria of the olive tree; because of some dietetic effect the bacteria seem to be essential to the life of the olive fly. In the Mediterranean region and in California the olive tree (*Olea europaea*) is often attacked by *Bacillus Savastanoi* which induces the formation of canker-like tumours or 'tubercles'.

These bacteria are spread by man through cuts and similar injuries but, under natural conditions, the olive fly (*Dacus oleae*) is the obligate vector. In the development of the larva from the egg to the pupal stage a large bacterial colony is found living continuously in the four blind ducts of the mid-gut (Fig. 116, B) and, indeed, these ducts are so arranged between the

peritrophica and the chitinous wall of the proventriculus (Fig. 117) that, even during ecdyses, the bacteria cannot be pushed out. The bacteria multiply unhindered and any excess passes out through the anus.

They persist throughout the histological and histogenic developments of the pupal stage and are found in the imago, e.g. in the pharyngeal glands.

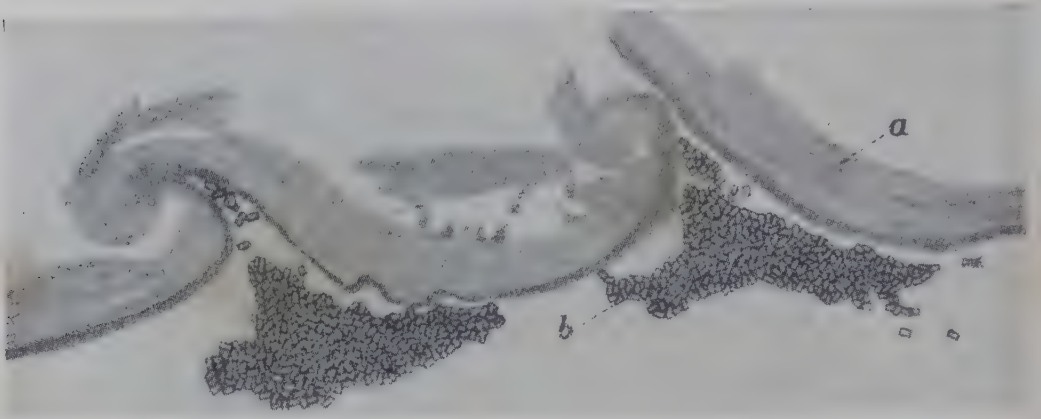


FIG. 114. Ascospores of *Ceratostomella ips* in the abdomen of a pine bark beetle (*Ips pini*). *a* skeleton, *b* ascospore masses. Approx. $\times 400$. (After Leach *et al.*, 1934.)



FIG. 115. Section through the alimentary canal of a pine bark beetle. *a* intestinal wall, *b* ascospores of *Ceratostomella ips*, *c* partially digested fragments of wood. Approx. $\times 400$. (After Leach *et al.*, 1934.)

From here they spread down into the mid-gut, and in the female they enter the anal glands situated just in front of the anus. The last abdominal segments are adapted to form an ovipositor (Fig. 118) through which the dorsal gut with its anal glands and the ventral vagina find a common outlet (Fig. 119); thus, persistent contamination of the eggs by bacteria is ensured.

In the adult insect the development of the bacteria, in contrast to that of the *Ophiostoma* (*Ceratostomella*) species mentioned above, is completely independent of the feeding habits of the vector but is governed by its

physiological condition, especially by the state of maturation of its sex organs. During oviposition (Fig. 120) a few bacteria are separated from the anal glands, reach the egg, multiply during the development of the embryo near the micropyle (Fig. 121), penetrate into the egg through the tracheae, and finally again colonize the proventriculus, thus completing the cycle.

The olive fly is, therefore, permanently and cyclically inhabited by *Bacillus Savastanoi*. It seems to obtain definite benefit as regards physiological growth substances from its guest, because the bacteria-free culture of embryos has so far failed. On the other hand, the olive fly harbours and feeds the bacteria and, as their vector, transmits them to new hosts where they again produce the well-known tumours. The life association between the bacteria and the vector is, therefore, an obligate one; without bacteria the vector cannot develop and, without the vector, there is no further spread of the bacteria.

The phylogenetic development of such mutualistic vital associations cannot be understood on the basis of these incidental pathogenic examples from plants, or animals, or man, but can only come from the study of the whole problem of animals and their endozoic micro-organisms.

Whereas contamination with *Ceratostomella* was indispensable to the life of the vector, and similarly, with *Bacillus Savastanoi* to the vector and the parasite, contamination with a plant pathogenic virus seems to be of vital importance only to the parasite. Most plant pathogenic viruses can be spread solely by insects and, under natural conditions, apart from direct vegetative transmission (p. 110), can usually pass from the distributor to the receiver only by means of the insects concerned. *Abutilon* mosaic is a striking example. In our glasshouses the leaf mottle of *Abutilon Thompsoni* does not spread from one plant to another but only from a diseased stock to a scion, whereas in the native country of the plant, the West Indies, the virus is transmitted directly from one individual to another by the indigenous insects (Bawden, 1939). The export of diseased stocks into other climatic regions where these insects are absent breaks the natural chain of infection.

Usually, it is leaf-sucking insects that function as vectors in virus trans-



FIG. 116. Semidiagrammatic longitudinal section through a mature larva of an olive fly. J mid-gut, S blind sacs of mid-gut, B colonies of *Bacillus Savastanoi*. (After Petri, 1909.)

mission (Storey, 1939), whether as alternate hosts (p. 87) or simply as carriers; it is the latter that interest us in this section. The transmission seems to be purely incidental, the insect sustaining neither visible benefit nor harm from it. In contrast to the insects serving as alternate hosts

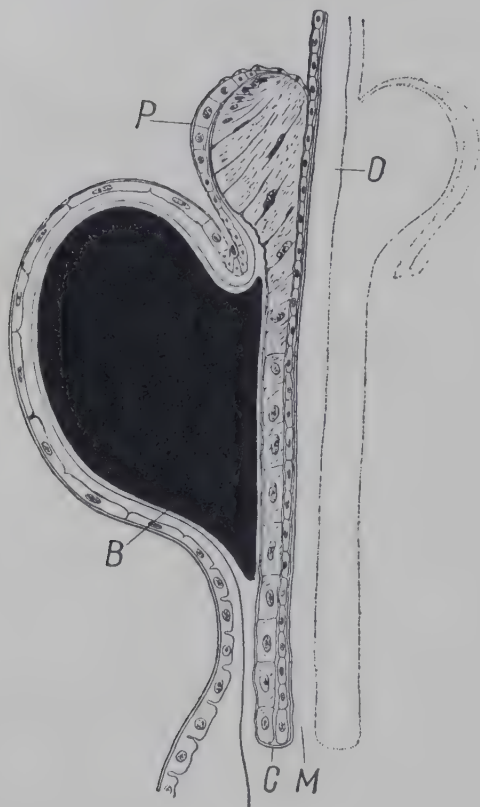


FIG. 117.

FIG. 117. Diagrammatic longitudinal section through the upper part of the digestive system of the olive fly. *O* oesophagus, *M* mid-gut, *C* chitinous layer of the proventriculus, *P* peritrophica, *B* colony of *Bacillus Savastanoi*. (After Petri, 1909.)

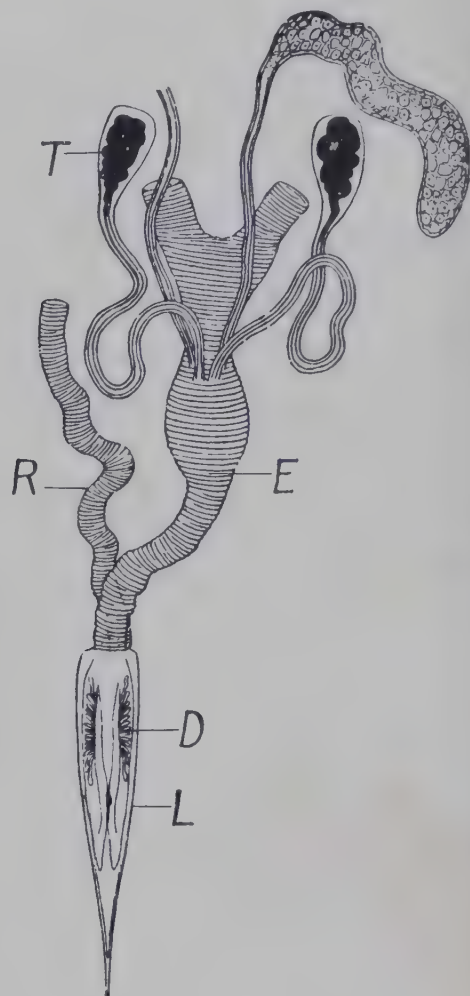


FIG. 118.

FIG. 118. Part of the female reproductive system of the olive fly. *T* ovary, *E* vagina, *R* hind gut, *D* anal glands containing bacteria, *L* ovipositor. (After Petri, 1909.)

(p. 88), those which are merely carriers can infect new hosts immediately after contamination, that is, without the interpolation of a circulation period (*Zirkulationszeit*); on the contrary, the sooner they alight on healthy tissues the greater are the chances of infection. Under certain circumstances the infectivity of the insect is lost after 12 hours and this fact can be used to separate the components of a mixed contamination of viruses in a vector.

The actual mechanism of virus transmission by the insect is unknown. It is difficult to think that the animals contaminate their mouth parts, as if they were a hollow needle, with virus-containing sap merely by sucking, that they then transport the virus epizootically and wipe it off on sound tissues when piercing them. This may occur in some cases, but in others

some endozoic mechanism must be involved in the transmission, although the insects are not definite alternate hosts (p. 87) and the virus undergoes no modification within them.

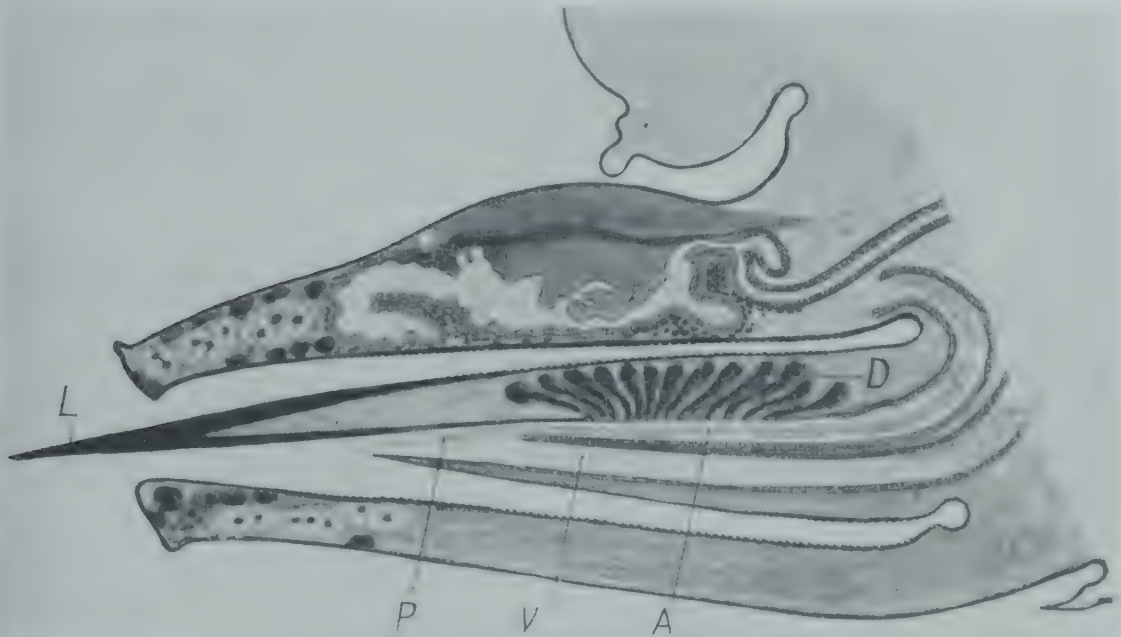


FIG. 119. Longitudinal section through the ovipositor of the olive fly. *L* lip of ovipositor, *D* anal glands with bacteria, *A* anus, *I* vagina, *P* peritrophica with bacteria oozing out. (After Petri, 1909.)

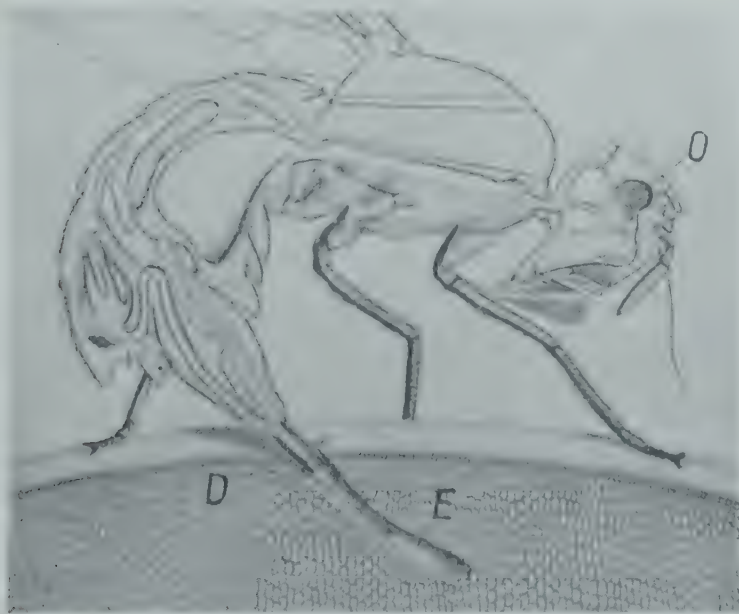


FIG. 120. An olive fly laying an infected egg. *O* diverticulum of oesophagus filled with bacteria, *D* anal glands with bacteria, *E* infected egg. Diagrammatic. (After Petri, 1909.)

This is indicated, in the first place, by the specialization of the insect vector. If the virus were simply transmitted hollow-needle-wise, then all sucking insects should be able to function equally well as vectors, except that those with larger mouth parts should receive more virus. This,

however, is not the case. Thus, the peach aphid (*Myzodes persicae*) is a more efficient vector of *Hyoscyamus* III virus than is the closely related *Myzodes circumflexus* (Watson, 1938).

Also those viruses which are readily inoculable in the laboratory by pricking with a needle should also be readily transmissible by insects, but this is not the case. Thus, potato virus X and tobacco mosaic virus can easily be transmitted by means of cell sap but not by *Myzodes persicae*. Therefore, the insect does not function merely as a hollow needle but inactivates these two viruses, which would scarcely be possible if they were simply adherent to the chitinous surface of the mouth parts.

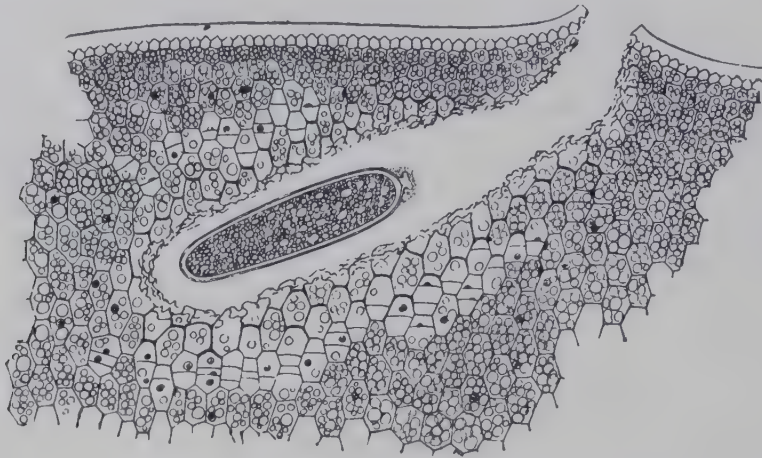


FIG. 121. Section through the outer tissues of an olive with an egg of the olive fly. At the micropylar end the olive knot bacteria are multiplying. (After Petri, 1909.)

Further, the infectivity of the insect vector is increased if it undergoes a period of fasting before it visits the virus-infected plant. Thus, if the aphides fast for 6 hours, are then allowed 2 minutes' feeding on tobacco plants infected with *Hyoscyamus* III virus, and are then transferred to healthy tobacco seedlings, an average of 72% of the latter becomes infected compared with 17% without preliminary fasting (Watson, 1938). As the aphides in both cases had fed equally long on the infected plants the contamination of their mouth parts must have been equally great, but their appetite was larger in the first case and they sucked up more virus-containing cell sap. If the contaminated insects are now made to fast, those that have already fasted lose their infectivity after 12 hours whereas the others lose it after only 1 hour: it would certainly be difficult to explain why the virus should adhere longer to the mouth parts of the fasted animal than to those of the others.

Finally, the reservoir character of the insect vector can be inferred from the evidence of a few attempts at single feedings (Watson, 1936). The probability of infection increases with the number of contaminated insects that feed on a plant, which is not surprising. In a particular experiment, when one aphid infected with *Hyoscyamus* III virus was placed on each of 100 healthy tobacco seedlings, 18% of the plants became infected, 5 aphides per plant gave 60% infection, 10 aphides gave 81%, and 20 gave 94%. But

the probability of infection also increases with the length of time during which the infected aphides are allowed to feed upon the healthy plants (Table XII). This would be incomprehensible if the insect vector merely wiped off its contaminated mouth parts on the healthy plant.

TABLE XII

The influence of duration of feeding of contaminated aphides on healthy tobacco plants: percentage of infection with Hyoscyamus III virus. (After Watson, 1936)

Duration of feeding of the contaminated aphides on healthy tobacco plants (hours)	Percentage of infected tobacco plants by the employment of (per plant)			
	1 aphid	5 aphides	10 aphides	20 aphides
	%	%	%	%
3	6.6	41.6	68.1	81.8
6	16.5	45.1	71.1	79.7
12	22.6	48.2	72.7	85.5
24	25.5	47.1	76.5	86.3
48	27.8	55.7	82.5	89.3

A similar decrease in infection, equally inexplicable by the hollow-needle theory, takes place when the one infected aphid is allowed to feed on two healthy tobacco plants one after the other. If it feeds on the first plant for only 2 or 5 minutes its infectivity on the second plant is undiminished, whereas if it is left on the first plant for more than 1 hour it does not as a rule infect the second plant (Watson, 1936). The decrease is true only for *Hyoscyamus III* virus and is not valid generally; in the case of sugar beet mosaic the one aphid, provided it is sufficiently contaminated, can infect four plants in succession (Watson, 1940).

Evidently the virus travels with the cell sap into the stomach of the insect vector and, later, during subsequent feeding, is released in the saliva. Despite this limited association the condition is one of simple internal contamination and not of an infection of the insect, since neither the insect nor the virus is altered or changed in any way as a result of the association.

(e) *Human Dispersal of Pathogens (Anthropochory)*

In human pathology, disease germs are most commonly transmitted by man himself; in central and western Europe almost the entire spread of infectious diseases is by human agency. Man is mobile and, as a distributor, he conveys the contagion himself to the receiver who admits it chiefly by inhaling droplets, or with his food, or through bodily contact. Thus, in contrast to plant pathology, it is not the pathogen which propagates itself from the distributor but it is the distributor who conveys it or passes it on.

In plant pathology, on the other hand, the human dispersal of germs plays only a limited role. In cases of direct transmission man's body itself is the vector; thus, *Bacterium michiganense* (bacterial wilt of tomato) is transmitted by the gardener on his finger-nails or his knife when pinching

out the tomato shoots. The same thing occurs with viruses and bacteria pathogenic to the potato when the seed tubers are cut.

More important, however, than direct transmission is the indirect transmission of plant diseases by man. In the course of his trade in seeds and plants he transports contaminated or infected plants or plant organs, and thereby creates new foci of infection which lead to outbreaks in new places, from which the disease can spread still farther. In this way smut and stripe diseases of cereals are spread by contaminated seed, and potato viruses, wart disease, and scab, &c., by infected tubers, from field to field and from one part of the country to another. The needle cast disease of the Scottish Douglas fir caused by *Rhabdocline pseudotsugae* was unknown south of the Main until a tree-nursery owner introduced it with plant material from Schleswig-Holstein into western Switzerland, from whence it spread rapidly (Terrier, 1942).

Indirect human dispersal of germs extends from continent to continent because of international trade. Thus, many of the most important infectious diseases of European cultivated plants derive from America or eastern Asia and were brought to Europe as a result of increased trading. Examples are late blight of potato (*Phytophthora infestans*), known in Europe since about 1830 and epidemic since 1845; powdery mildew of vine (*Uncinula necator*) noticed for the first time in England in 1845; blister rust of Weymouth pine (*Cronartium ribicola*) known in the Baltic States and Finland since about 1865; downy mildew of vine (*Plasmopara viticola*) first noticed in south-western France in 1878; American gooseberry mildew (*Sphaerotheca mors-uvae*) first noticed in western Russia in 1890, &c.

§ 4. The Epidemiology of Infectious Plant Diseases

If a disease affects single, isolated individuals within a population its occurrence is termed scattered or sporadic. If, on the other hand, it shows a concentration either in time or space it is known as a pestilence or an epidemic. Thus, epidemic connotes the frequent appearance and the local concentration of an infectious disease within a limited period of time.

When an epidemic is periodic, appearing after certain intervals of time, for example, if it flares up and dies down every year, it is termed cyclic.

When an epidemic has been established in a district for a long time it becomes endemic; e.g. black rust of cereals (*Puccinia graminis*) and red rot of pine (*Trametes radiciperda*) are endemic diseases in central Europe.

If, in a particular district, there is an outbreak of an endemic disease which affects a very large number of individuals, it may spread to a new region and the invasion of this new area acquires the character of a progressive epidemic or a spreading pestilence. If the epidemic extends over most of a continent and causes mass mortality, it is termed a pandemic.

Every epidemic runs a specific course; it changes in character, increases and becomes virulent, declines and becomes milder; it has its own

appearance and morphology. Hence section 1 deals with 'The Form of Epidemics'.

Typically, an epidemic is delimited in two ways; it has a beginning and an end, both in time and space. Hence section 2 deals with 'The Conditions for the Establishment of an Epidemic' (p. 140), and section 3 with 'The Conditions for the Decline of an Epidemic' (p. 170).

1. *The Form of Epidemics*

The current state of an epidemic is defined by statistical records. Quantitatively, the incidence of the disease, and qualitatively, the severity of the disease, provide the essential data.

In plant pathology the incidence or extensity of a disease is calculated from the number of diseased individuals in a given area or the number of lesions on a given test object; the severity or intensity of a disease is measured by the amount of damage caused; the mortality is the number of deaths in relation to the total number of individuals; and the lethality is the number of deaths in relation to the number of diseased individuals.

By graphic representation of the statistical data over a period of time epidemic curves may be constructed. The form of these allows two extreme types to be recognized: 'explosive' epidemics and 'tardive' epidemics. The curve of an explosive epidemic is characterized by a steep rise, a short acute peak, and a steep decline (e.g. Fig. 122). Tardive epidemics progress slowly, are long drawn out, and their curves do not show a paroxysmal peak (e.g. Fig. 123).

If the graphic representation of the statistical results is based on data extending over a calendar year, or over a growing season, it yields annual epidemic curves or annual cycles; with continuous data extending over a longer period it yields secular epidemic curves or secular cycles.

(a) *Annual Fluctuations of Epidemics*

In temperate climates, most plant epidemics in which the pathogen lacks a perennating mycelium, such as is present in the blister rust of Weymouth pine, run a characteristic seasonal course, or show an annual cycle or periodicity. They begin during the spring or summer, increase to a peak, and then diminish; the descending curve is rarely completed since it is terminated prematurely by harvest.

This rhythm in the seasonal appearance differs to a certain extent in different epidemics; the characteristic features are well known in practice but in only a few cases have they been proven by statistical data. The first isolated infections that initiate the outbreak of an epidemic in spring or early summer are usually overlooked, and the time at which they occur can only be surmised. The rise to the peak is more conspicuous. Under Swiss conditions yellow rust of cereals is usually already severe in June, potato blight in July, and leaf spot of sugar beet usually in autumn shortly before harvest. The annual curves for these epidemics thus show a single peak.

Fig. 122 illustrates this for the leaf spot of sugar beet (*Cercospora beticola*).

On 24 June the crown had 13 leaves of which 2 showed single lesions. By the end of July the disease was still very slight, the 25 leaves then present showing only 56 lesions. The curve then begins to steepen and after 28 August rises very sharply to a peak on 1 September with 12,940 lesions on the 37 leaves. From then on through the autumn there follows an equally sharp descent. The old heavily diseased leaves fell to the ground, and sometimes into the middle of the crown among the young leaves, yet these did not become diseased by the end of the experiment either because, in autumn, the necessary incubation time may be 10–12 days, or because the environmental conditions had become too unfavourable. Thus, on 19 September, at the time of harvest, the 8 youngest leaves out of the 21 still present are not

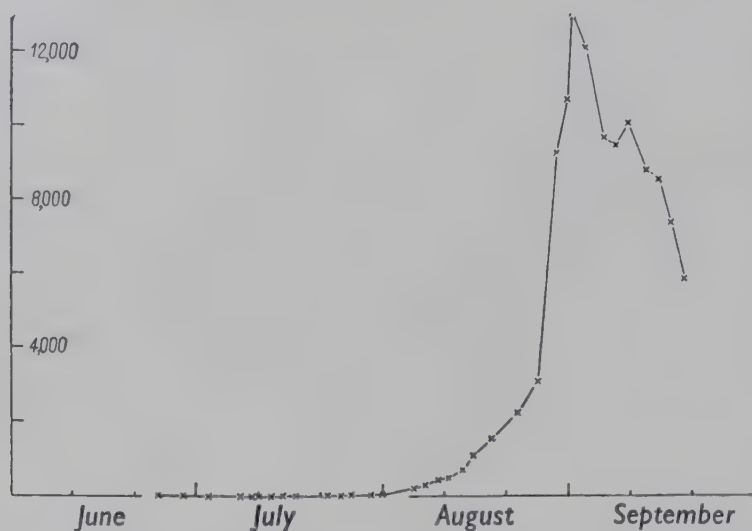


FIG. 122. The number of infection sites (leaf spots) caused by *Cercospora beticola* on the leaves of a single sugar beet plant from 24 June until 19 Sept. (After Pool and McKay, 1916.)

infected or bear only 1–3 lesions, in contrast to the 400–900 lesions on the older leaves. The right-hand side of the curve is approaching its end and the *Cercospora* epidemic is beginning to die away.

Annual rhythms are more complicated in those infectious diseases which, during their course, attack several organs (p. 82), and therefore develop in several waves. Thus, in apple scab (*Endostigme inaequalis*) the foliage, the fruit, and eventually the branches are attacked; in the downy mildew of vine (*Plasmopara viticola*), the foliage and the first buds, and in potato blight (*Phytophthora infestans*) the foliage and the tubers.

Apple scab may serve as an example. The annual epidemic starts in the months of April or May with the primary infections which result in leaf scab or early scab of the foliage (p. 89). From this it is spread by numerous generations of conidia until, in the autumn, nearly all the leaves on the tree are infected, giving secondary infections or late scab of the foliage. A premature leaf-fall takes place which reduces the assimilating leaf area. If the number of new infections of leaf scab are depicted graphically a saddle-shaped curve similar to that in Fig. 122 is produced.

Immediately after flowering a new wave of infection sets in on the young

fruits (early scab of the fruit, Fig. 86), the spores being derived from the late scab on the foliage. This spreads steadily until harvest (Fig. 88) so that occasionally, under natural conditions and in the absence of control measures, 100% of the fruits become infected (late scab of the fruit). The rhythm of this second epidemic wave, that of fruit scab, develops in a completely different way to that of leaf scab (see Fig. 123), namely, with a continuous increase.

After harvest and during transport or storage a third epidemic wave begins, that of storage scab (Fig. 89), a secondary infection, which arises

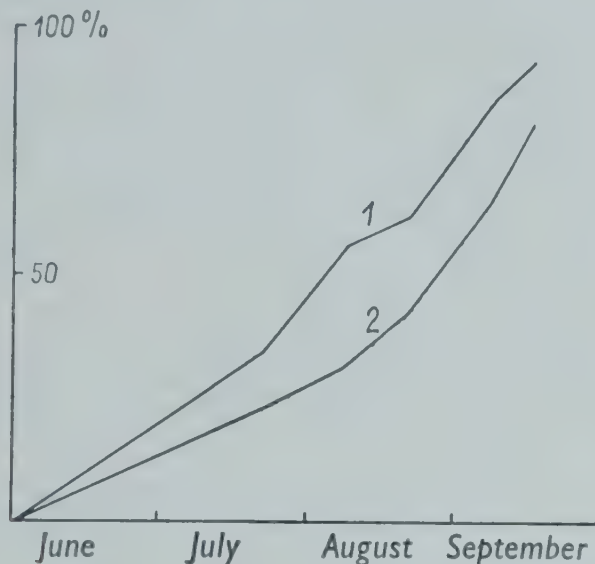


FIG. 123. The increase in scab attack on apples in the course of a growing season. Ordinates: % disease on fruit of Golden Pearmain (curve 1) and on Cox's Orange (curve 2). (After K  the, 1937.)

from scabbed fruit and causes severe economic loss. Its rhythm differs completely from the two other annual rhythms and, in contrast to the *Cercospora* example, it is not completed with harvest but only begins then.

Accurate knowledge of these three annual rhythms forms the basis for the chemical control of apple scab. If it is successful in controlling the infection chain of early scab on the foliage, i.e. the primary infection, late scab is consequently much reduced. Without early scab on the foliage leaves there is no late scab and, in the same way, without late scab on the fruits there is no storage scab, &c.

(b) *Secular Fluctuations of Epidemics*

The secular or long-term periodicity of progressive epidemics differs from that of endemic epidemics.

Progressive epidemics that have recently been introduced into a region usually develop bilaterally. During the first few years they increase destructively, but later settle down to a constant value.

For example, a secular course of this kind was followed in the colonization of the European vine-growing areas by the powdery mildew of

vine (*Uncinula necator*, = *Oidium Tuckeri*). The fungus derived from North America or eastern Asia (Japan) and was first recorded with certainty in England in 1845. It immediately spread to the Continent where it produced a true epidemic. In 1848 it was established only in the vicinity of Paris, but by 1851 it had spread to southern France, Italy, southern Tyrol, Switzerland, and western Germany. In the following year it occurred in North Africa (including Madeira), and the eastern Mediterranean countries, in 1866 in Australia, later in South Africa, and in 1891 in Brazil. To-day it occurs in all vine-growing areas.

The critical years were 1848–52, during which it literally exploded in the vine regions of Europe and North Africa. During this time it not only colonized an enormous area but it exhibited an immense pathogenicity. During the years 1850–4 the average yield of wine for the whole of France, i.e. including both infected and non-infected areas, fell to under a quarter (Table XIII), namely, from 20·7 hl. per hectare to 4·9 hl., an economic disaster for the whole country.

TABLE XIII

The decrease in yield of wine in France resulting from the invasion by powdery mildew (Uncinula necator). (After Müller, 1918)

Year	Yield of wine	
	Total (millions of hl.)	per hectare (hl.)
1850	45·3	20·7
1851	39·4	18·1
1852	28·6	13·2
1853	22·7	10·4
1854	10·8	4·9

In 1854, some 6 years after its introduction, the mildew epidemic had passed its peak and during the following years it declined and reached comparative equilibrium, a steady value. It became endemic in France and in all the chief vine-growing areas, although in a milder form so that it could be controlled by chemical methods. This decline was due not only to external factors, e.g. the therapeutic use of sulphur-containing materials, but undoubtedly also to internal causes, which will be discussed in section 3 ('The Conditions for the Decline of an Epidemic').

A comparable secular periodicity, i.e. an alarming outbreak in a new area, the increase of the damaging effect to a peak, and a later amelioration of the epidemic to a steady value, occurred with potato blight (*Phytophthora infestans*). From contemporary accounts it may be presumed that 100 years ago, i.e. at the beginning of its appearance in epidemic form (p. 134), it was much more virulent than it is to-day. In Ireland it brought a famine in the course of which about a quarter of a million people starved to death. One reads in the contemporary account by Jeremias Gotthelf about grandmother Käthi (Eugen Rentsch Verlag, Erlenbach, p. 117 et seq.). The grandmother having heard in the village of the new epidemic on the

potato, hurried home and in the evening went out into the field; in one hand she held a flickering lantern and in the other her grandson Johannesli: 'now by the light of the lamp Käthi saw the dreadful black pestilence on all her potatoes. As she shone the lamp on them, the pestilence became blacker and more dreadful. Then misery overcame the old woman. She sat down in the furrow and wept bitterly and Johannesli sat down beside her and wept still more bitterly, not because of the potatoes but because his grandmother wept so bitterly and would not tell him why.'

The damage was vastly increased by the uncertainty as to whether one could eat the attacked potatoes without catching the 'plague'. 'The potatoes

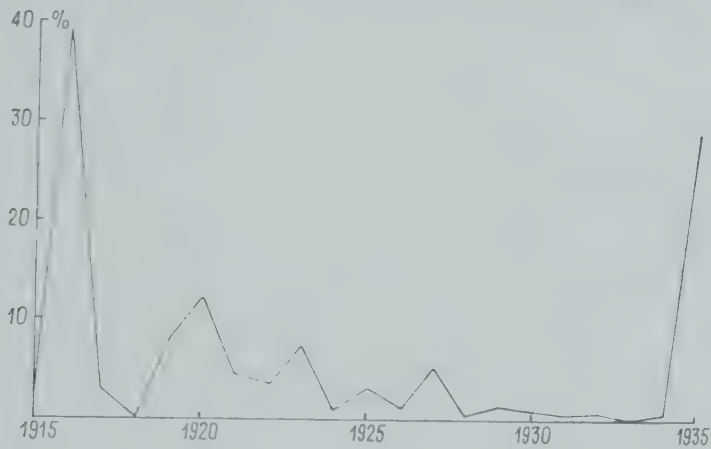


FIG. 124. The probable loss in yield of wheat (spring and winter) caused by black rust (*Puccinia graminis tritici*) in the chief wheat growing areas of the United States from 1915 to 1935. (After Stevens and Wood, 1937.)

have pestilence spots and, therefore, whoever eats them, man or beast, must die.' To-day the loss in yield due to *Phytophthora*, although still important, rarely amounts to more than 20–30%.

An exception to this cyclic development of progressive epidemics is given by the downy mildew of vine (*Plasmopara viticola*) which was introduced into south-western France in 1878 and since then, just as in the case of the powdery mildew, has spread throughout the Continent. Year after year, however, so far as climatic conditions permit, it occurs with the same severity. This epidemic has, as yet, shown no diminution in intensity and, therefore, we still do not know whether the descending branch of the curve will be manifested in the course of time.

A complete contrast to the progressive epidemics so far discussed, which break out in new areas, is shown by the secular behaviour of those diseases which are indigenous or endemic in a region and recur year after year in a mild or severe form. In this case there is no secular rhythm of increase and decrease and the endemic disease appears, apart from meteorologically conditioned fluctuations, to have reached a steady value.

Fig. 124 shows the probable loss in yield caused by black rust (*Puccinia graminis tritici*) in the years 1915–35 in the chief wheat-growing areas of the United States of America. The curve does not exhibit a definite

secular periodicity but merely the effect of favourable or unfavourable weather conditions during those years (see Fig. 125).

The severity of endemic infectious plant diseases, if one considers disease in a continent as a whole, does not appreciably change in historical time, it is only its economic importance that continues to increase.

2. *The Conditions for the Establishment of an Epidemic*

What are the conditions under which an infection gives rise to an epidemic? For a source of infection to expand to epidemic proportions a number of conditions must obtain *simultaneously* in both the pathogen and the environment, in particular, there must exist:

on the part of the host, an abundant supply of susceptible individuals produced by

- (a) an accumulation of susceptible individuals (p. 140);
- (b) heightened disease proneness of the hosts (p. 141);
- (c) the presence of appropriate alternate hosts (p. 144);

on the part of the pathogen, the possession of high infective capacity, i.e. a high epidemic potential, conditioned by

- (d) the presence of an aggressive pathogen (p. 148);
- (e) high reproductive capacity (p. 154);
- (f) efficient dispersal (p. 157);
- (g) unexacting growth requirements (euryzoic) (p. 160);

on the part of the environment

- (h) optimal weather conditions for the development of the pathogen (meteoropathology, p. 160).

The chief difficulty in the establishment of an epidemic lies in the synchronization of these conditions. All these eight groups of factors must be realized in a particular locality at one and the same time, and if any one of them be delayed the progress of the epidemic becomes doubtful. Therefore, the number of epidemiogenic parasites is very limited compared to the total number of parasitic micro-organisms.

These eight factor groups and their consequences will be discussed briefly in turn, and finally, in a ninth section, the reason for the increase in the severity of an epidemic whilst its curve is rising will be considered.

(a) *An Accumulation of Susceptible Individuals as a Condition for an Epidemic*

With increasing distance from the distributor the probability of infection owing to germ dispersal falls off rapidly (Fig. 106). Presumably, therefore, an epidemic would rarely break out in host plants that occur only as single isolated individuals in the open. Experience with *Puccinia Komarovi* and *P. malvacearum* shows, however, that the rate of dissemination or migration of a pathogen can be astonishingly high even on wild plants.

The rust fungus *Puccinia Komarovi* lives on the small yellow balsam

(*Impatiens parviflora*), a weed of roadsides and rubbish dumps, and on some other *Impatiens* spp. It was described by Tranzschel in 1904 from central Asia (Turkestan, Dsungarei, Altai) and the northern Himalayas. It was probably during the First World War that it escaped from this area and spread to the West. Within 5 years it had spread through central Europe, reaching the Ukraine in 1921, Mark Brandenburg in 1933, Silesia, Hesse, Baden, and Bavaria in 1935, Switzerland in 1936, and Hungary in 1938; and in these areas it followed its host to the most remote corners. Thus, the author found it in 1941 in an isolated forest ravine in Domleschg in the Grisons!

Puccinia malvacearum which is found on Malvaceae, especially species of *Malva* and *Althaea*, derives from Chile and appeared for the first time on the European continent in Spain in 1869. In 1873 it was recorded in France, and by 1874 in England, Denmark, Germany, and Italy. Thus, this parasite, which attacks only wild and garden plants, succeeded in invading the whole of western and central Europe in the course of 5 years.

It is clear, therefore, that given a sufficient reproductive capacity in a pathogen its rate of dissemination and migration on plants growing singly must not be underestimated.

How much greater, therefore, must be the danger of an epidemic where, as in agricultural mass culture or monoculture, vast areas are thickly planted with the same susceptible host species. Thus, the wheat-growing regions of North America extend in one united tract from southern Mexico to northern Canada, a distance of about 3,000 km. without any natural barriers; the rice area in south-eastern Asia is another example. For economic reasons there occurs in these areas an accumulation of susceptible individuals, and in consequence the possibility of a concentration of germ dispersal and disease which, in human medicine, is never reached even in the most densely crowded places, such as large towns or military camps.

(b) Heightened Disease Proneness of Hosts as a Condition for an Epidemic

Although an epidemic can break out even with an average disease proneness in individual hosts, its development is promoted by a heightened disease proneness which may be due either to innate or to environmental factors.

Ontogenetic variation in disease proneness (see Chapter 4), e.g., the transition from youth-resistance to age-susceptibility, is innately determined. On occasion the susceptibility of potato plants to blight (*Phytophthora infestans*), of plum leaves to plum rust (*Tranzschelia pruni spinosae*) of apple leaves to scab (*Endostigme inaequalis*), and of rose leaves to black spot (*Diplocarpon rosae*) increases with the age of the plant and with the growing season. Thus, the resistance correspondingly diminishes during the vegetative period, and therefore, so far as the host is concerned, conditions as a rule favour these epidemics more at the beginning of September than at the beginning of June.

On the other hand the resistance of peach trees to leaf curl (p. 93) increases during the vegetative period.

In temperate climates the ontogenetic variations of disease proneness are repeated annually in the same rhythm and one speaks, therefore, of a cyclic disposition of the host to the particular epidemic.

The environmental factors which heighten disease proneness can be arranged epidemiologically in three groups, the susceptibility of the host being increased by (a) a general lowering of its vitality, (b) the external temperature, and (c) nutrition. These will be discussed fully in Chapter 4.

The epidemic twig blight of Arolla pine (*Pinus Cembra*), the Swiss mountain pine (*P. montana*), and the Austrian pine (*P. austriaca*), which occurs in afforestation schemes, is conditioned by various factors, one being a general lowering of the vitality of the host.

In autumn the ascospores or the conidia of the pathogen, *Crumenula abietina*, reach the base of the apical buds of the twigs, the most usual site of infection. The mycelium grows in the infected buds during the dormant winter period, kills them, and penetrates through the bark into the twigs. In course of time it enters the bases of all the needles so that, in the following spring, these become characteristically browned from below upwards, and finally the tree dies (Ettlinger, 1945).

The disease also occurs generally in nature but is unimportant. The pathogen is ubiquitous.

Twig blight is known as a devastating malady only in afforestation schemes such, for example, as the black pine plantations of the Danish and Norwegian heaths and dunes in the eighties of last century, and in more recent times, 1936–40, in Alpine plantations in Albertitobel above Davos. The local increase in the incidence of the disease and of its destructive course must, therefore, be associated with definite conditions and primarily with a lowered vitality of the transplanted trees. In the centuries between clearing and afforestation the soils retrogressed, degenerated, and were leached out until they were of no use for pasturage; then they were handed back to the forester to make them into 'forests'.

In such degenerate soils every kind of tree is unsuitable even if carefully chosen because the soils themselves are no longer forest soils. Because of these soil deficiencies, the competition of the sparse weeds, and the abnormal local climatic conditions, the vitality of the transplanted trees is lowered to such an extent that there is a heightened disposition to attack and disease by *Crumenula*.

This disposition due to the site is accompanied by conditions favourable to the development of the pathogen. Usually the latter spreads into the host during the dormant period, and is, therefore, particularly dangerous to snow-covered branches and individuals. Outside the snow cover the winter temperatures are too low for the pathogen. These conditions are precisely those fulfilled in young (less than 'man high') plantations over extensive tracts.

Thirdly, every afforestation scheme of necessity creates a local accumulation of individuals of the same age; consequently the depression of vitality

reaches the critical value throughout entire stands at about the same time. This gives rise to a local concentration of susceptible and diseased individuals which leads, in turn, to an increase in both the germ density and the infection density, which results in the well-known devastating course of the malady.

When these three epidemiological conditions coincide the pathogen spreads from its natural habitat to plantations. It loses its harmless character and gives rise to a local non-progressive epidemic. But the local restriction of the predisposing factors does not allow the epidemic, in spite of its alarming appearance, to spread back appreciably to natural woodlands in the vicinity.

A like situation, an ubiquitous pathogen on the one hand and a locally restricted disposition of the host on the other, is found in many infectious diseases of man and other animals. In spite of the epidemic capacity of a pathogen a progressive epidemic does not always ensue. Thus the pathogen of the nosema disease of bees, the sporozoan *Nosema apis*, which lives in the epithelial lining of the gut, is always present in bee communities, yet a depression in the vitality of the bee population must occur before it can become really pathogenic (Morgenthaler, 1944).

A cyclic increase of proneness to an epidemic due to the external temperature is found, for example, in black rust of cereals. The annual epidemic curves show a yearly increase of the black rust epidemic up to mid-summer; the resistance of cereal plants to black rust generally decreases with increasing temperature.

In the secular epidemic curves warm years usually appear as black rust years because of this relation to temperature. The horizontal axis running through the middle of Fig. 125 represents the mean temperature for the three principal growing months in the summer-wheat areas of the United States. The black columns represent severe rust years and the white ones slight rust years; the length of the columns is proportional to the incidence of the rust attack. In general, high average temperatures correspond to severe epidemics of black rust and low temperatures to slight ones; as a rule, an epidemic of black rust does not occur when the mean temperature is below 16.5°C .

It might be expected that increases in proneness to epidemics in cultivated plants due to nutrition would result from the application of chemical fertilizers, but this has never been proved. The disease proneness of an individual plant or an individual crop is (as we shall see in Chapter 4) undoubtedly affected by unbalanced nutrition, but such individual cases are insignificant in the epidemiology of a whole country or continent.

Conjectures on this subject have possibly been influenced by a false analogy to the conditions found in man. Thus, in man, the seasonally conditioned vitamin deficiency of early spring can give rise to a cyclic disposition, e.g. to influenza. Again, in time of war the nutritional status of the majority of the individuals of a whole country or region can sink below the pathogenic threshold so that a nutritionally governed dis-

position to certain epidemics is produced in these countries or regions; hence, the post-war spread of epidemics.

In plants, however, the conditions are such that the basic food requirements are guaranteed by the soil from the beginning. The only problem for discussion here is the additional nutriment given with the aim of raising the yield; this, for economic reasons alone, can be unbalanced or excessive only in single cases which, therefore, do not matter from an epidemiological point of view. Hence, the idea that cultivated plants may become more

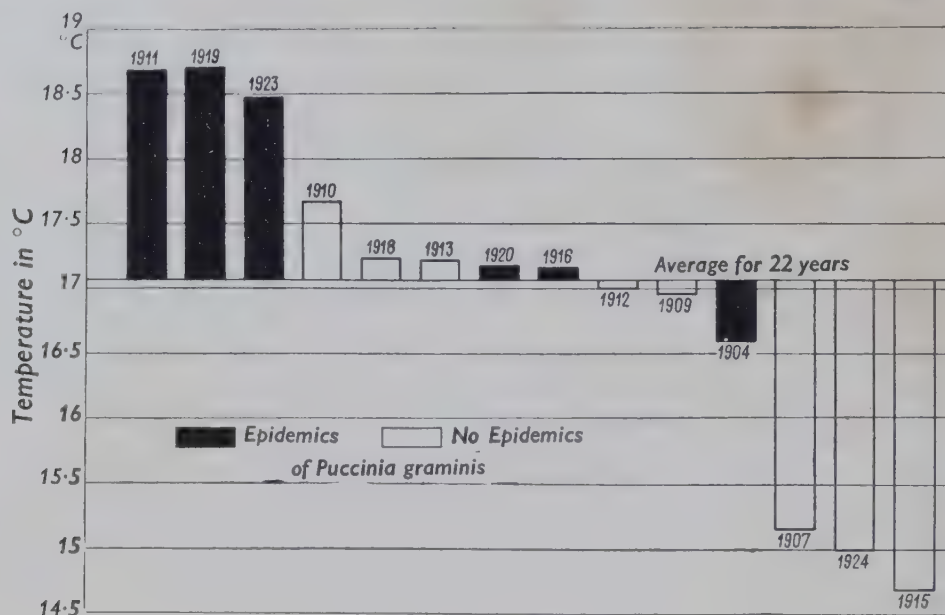


FIG. 125. Relation between the mean temperature for the months May, June, and July in the principal spring-wheat-growing States of the Union (Minnesota, North and South Dakota) and the epidemics of black rust occurring from 1904 to 1925. (After Stakman and Lambert from Fischer and Gäumann, 1929.)

susceptible to epidemics because of manuring with artificial fertilizers is misleading.

(c) *The Presence of Appropriate Alternate Hosts as a Condition for an Epidemic*

The presence of the appropriate alternate hosts is a condition for an epidemic only in those diseases with heterogeneous infection cycles (p. 83); in particular for obligate host-changing rust fungi (example (aa), cluster cup rust of pear trees) and viruses (example (bb), leaf roll of potato).

(aa) In cluster cup rust of pear (*Gymnosporangium Sabinae*, p. 86), the immediate presence of the alternate host, *Juniperus Sabina*, &c. within at most a radius of 100 m. is the *conditio sine qua non* for a local epidemic. If junipers are eliminated the epidemic disease comes to an end and, therefore, one of the basic rules in fruit-growing is the destruction of infected juniper bushes.

(bb) Where potatoes with leaf roll (p. 86) are growing in aphid-free districts, e.g. on wind-swept coasts and mountain sides, the transmission of the virus from one plant to another does not take place; therefore, no

epidemic of leaf roll occurs, any more than a malaria epidemic can break out in the absence of *Anopheles*. In cases where the grower eliminates the diseased plants by rogueing, he breaks the cyclic transmission of the virus from the mother plant to the tuber and the crop remains free from leaf roll. This is one of the principles applied in the raising of potato seed free from leaf roll.

In an epidemic with a facultative homogeneous infection chain the direct result of action against the alternate host is naturally less effective than in the case of cluster cup rust of pear. Thus, two of the most important cereal rusts, black rust (*Puccinia graminis*) and crown rust (*P. coronata* and *P. lolii*), overwinter, at least in central Europe, mainly by teleutospores and, in the regions of this discontinuous infection chain, pass over the following spring to the appropriate hosts, namely, *Berberis vulgaris* for black rust and *Rhamnus Frangula* and *R. cathartica* for crown rust. But in the absence of these bushes, or if they have been uprooted, the increase of these epidemics is merely postponed and not prevented as in cluster cup rust.

Both rusts produce asexual spores (uredospores), and thus they can maintain themselves without a change of host. Their dikaryotic mycelium overwinters on the autumn-sown crop (p. 91) and wild grasses from which, in the following spring, the infection cycle proceeds continuously in the cereal fields. The immediate result of measures for uprooting barberry bushes, &c., depends, therefore, to a large extent on the local existence of such vegetative infection chains. Nevertheless, the elimination of the alternate host is always justifiable particularly in view of an indirect effect: in the absence of the alternate host hybridization, which will be discussed later, does not occur in these parasites.

In plant pathology there are also records of the contrary procedure when, for economic reasons, a highly susceptible alternate host has been newly planted in some area and thereby the conditions for the breaking out of a hitherto endemic rust disease have been created. An instructive example is given by the blister rust of Weymouth pine caused by *Cronartium ribicola*.

Its haploid phase develops on five-leaved species of *Pinus* and forms pycnidia and aecidia on their trunks giving blister rust, especially of the Weymouth pine. The dikaryotic phase inhabits species of *Ribes* and produces pustules of uredospores and teleutospores on their leaves, giving the leaf rust of currants, &c. Its development corresponds to that of *Puccinia graminis* (p. 84). In early summer its aecidiospores infect young *Ribes* leaves, and during the summer its uredospores spread it to surrounding *Ribes* bushes on which, in the autumn, the winter spores (teleutospores) are formed. In the following spring the latter produce basidiospores which infect five-leaved species of *Pinus* from which, 2 years later, the cycle again begins on the *Ribes* bushes. The fungus can spread from *Ribes* to *Ribes* during the vegetative period but not from *Pinus* to *Pinus*; from *Pinus* it must infect *Ribes*, and from *Ribes*, after the production of winter spores,

it must pass over to five-leaved pines. Hence, the alternation of hosts is facultative except at the start and the end of the vegetative period, when it is obligatory. Consequently blister rust can only occur where both hosts are found together.

In nature it is endemic in the Alps and in east Russia and Siberia (Fig. 126, *A* and *S*) and alternates there between the Arolla pine (*Pinus Cembra*) and wild *Ribes* species. The existence of these two geographically separated or disjointed areas confirms the rule mentioned on page 88, that heteroecious parasites usually only migrate with their plant associates. The Late Tertiary northern Arolla pine reached the Alps during the course of the Ice Age, and after the retreat of the glaciers formed a relict area there in which the blister rust was also able to survive. In this way there arose in Eurasia the two well-known blister rust areas, the Alpine and the east Russo-Siberian. The economic importance of the Alpine area has been, as yet, very slight because of the high resistance of the alpine races of the Arolla pine, whereas the Siberian race appears to be relatively susceptible.

The later epidemics of blister rust were brought about by the combined effects of natural and man-made conditions.

The natural conditions are such that the vast European region which separates the two foci of blister rust from one another has, since the Ice Age, contained numerous wild and cultivated *Ribes* species such as *R. Grossularia* (gooseberry), *R. nigrum* (black currant), and *R. rubrum* (red currant). Yet blister rust could not enter this region because, after the *Ribes* bushes have shed their leaves, no overwintering by means of uredospores is possible, i.e. there could be no persistent homogeneous infection chain. Further, the alternate host for the heterogeneous infection chain, the susceptible five-leaved *Pinus* spp., was absent.

On the other hand, both conditions for the epidemic, i.e. the presence of susceptible five-leaved pines, and a coincident area of *Ribes* bushes, were present together across the ocean in the northern United States and Canada. Yet there the parasite was absent, and consequently there was no disease.

Man, with his economic activities, now interfered with this unstable balance in the European and the North American-Canadian areas.

Let us first discuss the European area. The foresters imported the Weymouth pine (*Pinus Strobus*) from the eastern United States because of its soft, knot-free wood and its rapid growth of valuable timber, and they planted it on a vast scale at the beginning of the eighteenth century throughout the whole of Europe north of the Alps. They thought themselves all the more justified because, in its homeland, the Weymouth pine had no serious enemies other than the honey agaric. Because of these plantations in Europe north of the Alps a new association of five-leaved *Pinus* spp. with *Ribes* bushes was artificially created so that now four such areas existed:

1. the natural Alpine Arolla pine-*Ribes* area with endemic blister rust;
2. the east Russo-Siberian Arolla pine-*Ribes* area, also with endemic blister rust;

3. the natural North American Weymouth pine *Ribes* area still without blister rust; and
4. the man-made central and north European Weymouth pine *Ribes* area also as yet without blister rust.

The artificial production of a five-leaved *Pinus-Ribes* area in the immediate vicinity of the endemic Alpine and east Russo-Siberian disease area created a grave latent danger for the Weymouth pine throughout the entire northern hemisphere, but as yet the pathogen was absent from the Weymouth pine areas and with it the disease.



FIG. 126. Epidemiological distribution of blister rust of Weymouth pine. *A* and *S* endemic Alpine and Russo-Siberian blister rust area of the *Arrolla* pine. *N* newly created North American blister rust belt in the natural *Pinus-Ribes* area. Original.

The disease could first appear epidemically after the European *Pinus Strobus* area had been completed and joined up with the east Russo-Siberian blister rust area, a position that was reached after nearly 100 years. As a result, the rust attacked the Weymouth pine, a new host on which it had not previously been recorded. The epidemic area, which is not shown in Fig. 126, was extended to the west; it reached the Baltic States in 1865, caused epidemics in Finland, Denmark, and Germany between 1865 and 1879, in Sweden in 1880, in Holland in 1885, in France in 1889, in Great Britain in 1892, in Belgium in 1894, and in Switzerland in 1895. The attack was so devastating in these planted areas that, in the first few years, *Pinus Strobus* seemed to be doomed.

It is noteworthy that the epidemic did not derive from the Alps, which are geographically nearer, but from the more distant east Russo-Siberian focus. The slight intensity and severity of the disease in the Alpine *Pinus Cembra* area, the gap of about 30 km. as the crow flies between this and the central European *Pinus Strobus* area, and the height of the mountain wall, obviously formed an insurmountable barrier for the completion of the infection chain between the endemic Alpine *Pinus Cembra-Ribes* area and the newly produced central European *Pinus Strobus-Ribes* area.

In the European *Pinus Strobus*-*Ribes* area the pathogen attacks Weymouth pine and currant bushes equally. The name and importance of the epidemic depends on the occupational point of view; the forester speaks of the Weymouth pine blister rust and blames the owner of currant bushes who provides the alternate host, whereas the gardener speaks of currant rust and blames the forester for this gift.

Within 30 years the blister rust had spread epidemically throughout the artificially produced European *Pinus Strobus* area, and in 1909 it was introduced with young trees into the northern United States. It invaded not only the artificially created area but also the natural *Pinus Strobus*-*Ribes* area in the native habitat of these trees. Here it found *Pinus Strobus* (eastern white pine) in the east of the continent, and *Pinus monticola* (western white pine) in the west, together with other wild, susceptible, five-leaved *Pinus* spp., *P. flexilis*, *P. parviflora*, &c. The fungus had thus at its disposal an uninterrupted wild stand, and in consequence the blister rust epidemic in North America swelled into a pandemic (Fig. 126, N).

Since 1922 the suppression of the disease has been attempted by the wholesale destruction of all susceptible *Ribes* bushes in the natural distribution area of *Pinus Strobus* and *Pinus monticola* (see Fig. 126) and by a systematic elimination of all infected trees.

The example of blister rust of Weymouth pine shows how a harmless, smouldering endemic disease can grow into a world-wide and destructive pandemic causing huge damage when a susceptible alternate host is introduced into a latently dangerous region, in this case a natural *Ribes* area.

In this example it is important, from the epidemiological point of view, to note that the blister rust on Weymouth pine was brought from North America to Europe by the import of *Pinus Strobus* and was easily taken back again from its old habitat, Europe, into a new area, North America. By the mass planting of a susceptible alternate host in the dangerous European *Ribes* area, man thus completed a new infection chain which formed a bridge from the east Russo-Siberian relict focus to the west, and round the world to the eastern and, later, to the western coasts of North America.

It is clear that experiences of this kind are arguments not only against the import of foreign species of plants but also against the export of our own plants.

(d) *The Presence of an Aggressive Pathogen as a Condition for an Epidemic*

Infection is an essential condition for an epidemic, and in the absence of germs no infectious disease or epidemic can break out even when all other conditions are fulfilled. But the germ is only a condition, not the cause of an epidemic. An epidemic occurs when the eight factors mentioned on page 140 coincide in time and place, i.e. when the whole group of causal factors is realized at the same time in a particular district.

The condition that an aggressive pathogen must be present in a particular region can be fulfilled epidemically in five ways:

(aa) Because of its historical development the flora of a given region may not contain a particular pathogen, but this may be introduced.

Specialized parasites are, as a rule, geologically younger than their hosts. When a unitary Tertiary floristic area such as the Euro-Siberian-North American area breaks apart for geological reasons, collapse of land bridges, &c., into two separated floristic regions, not only the development of the phanerogamic hosts but also the differentiation of their parasites follow their own courses in these new regions.

Within the phanerogamic host flora the later independent development leads to the formation of numerous substitutive species which are closely related biologically and systematically to the corresponding species in the other floristic area. But since the separation of the regions they have evolved independently and have become saturated by the pathogens which have originated in the new regions since their geological separation. Selection by the pathogens has prevented the highly susceptible individuals from reproducing themselves further and has thereby eliminated the corresponding genetic factors. In its endemic habitat every epidemic is, therefore, relatively mild.

If a pathogen, presumed to have arisen after the separation of the two floristic regions, be transferred into the other region it brings about on the substitutive species within a few decades a disease saturation which, in its original home, would have needed geological periods of time. Hence, the course of the epidemic due to such an adventitious pathogen may, during the first decade, attain a severity which would never have been suspected from observations in the old-established floristic region.

It is thus not an accident but an outcome of floristic evolution that during the last century Europe has received the pathogens of some epidemics from North America, and that North America has derived some of its pandemics from Europe. In both cases it may be assumed that the parasites involved first originated after the breaking up of the Euro-Siberian-North American floristic area, and therefore that they have since had no opportunity to select out the highly susceptible relatives of the substitutive species.

Two examples will be given of attack in each direction, North America to Europe and Europe to North America.

In North America the genus *Vitis* comprises many species which are obviously saturated with two native parasites, powdery mildew (*Uncinula necator*) and downy mildew (*Plasmopara viticola*), and to-day the vines are, therefore, only relatively mildly susceptible to both. Although it might be suggested that by accident all the species in nature had become highly resistant, it would be very difficult to sustain this hypothesis. Europe possesses a single species, *Vitis vinifera*, the grape vine, which was free from the two North American parasites, but when these were brought into Europe during the nineteenth century (pp. 137 and 139), saturation occurred in the form of the well-known epidemic.

The gooseberry mildew (*Sphaerotheca mors-uvæ*) has been known for

over a hundred years in North America as a dangerous *Ribes* disease, preventing the cultivation of the best European gooseberries, which are destroyed by it. After its introduction into Europe towards the end of last century, the disease, previously unknown on the gooseberry population of Europe, again assumed a disastrously epidemic form.

The converse development, harmless endemic parasites in the disease-saturated Old World and fulminant new pathogens in the unsaturated hosts of the New World, has been illustrated by the Weymouth pine blister rust (*Cronartium ribicola*), mentioned above.

A second example is provided by the Ascomycete *Endothia parasitica*, which lives on Mediterranean and eastern Asiatic Cupuliferae. Evidently it has saturated these from ancient times, and therefore causes only a relatively harmless chestnut blight, for instance on the Spanish chestnut (*Castanea sativa*). About 1904 it was introduced into North America and spread to both wild and cultivated species of *Castanea*, and to some *Quercus* spp., in the form of an uncontrollable pandemic, the chestnut bark canker, which devastated great forest tracts and caused untold damage. It is likely that during this pandemic more aggressive and virulent strains originated, but the pandemic would not have broken out as it did if the pathogen had been able during geological time to saturate the *Castanea* stocks of the New World as had occurred in the Old World.

In view of these events the question arises, what further epidemics may be expected in Europe on account of this kind of floristic evolutionary differentiation and consequently what quarantine measures, &c., must be adopted? Prediction is not easy because the endemic diseases usually take on a harmless character in their disease-saturated home regions and only regain their original pathogenicity after the outbreak.

Nevertheless, it must be recognized that there exists an immediate latent danger for Europe in an epidemic which is already causing serious damage in North America; this is the fire blight of the Pomoideae, especially apple and pear, caused by *Bacillus amylovorus* (p. 49).

(bb) The distribution of a cultivated plant may be extended artificially by world trade and thus, for a time, its native parasites may be left behind; however, they follow later.

Useful agricultural plants and forest trees are generally introduced into new cultivation areas by seed and more rarely by vegetative organs, tubers, &c. The danger that the parasite will be spread with their first growth is slight, since only the best and healthiest seed is used for such purposes. In this way the new region of cultivation becomes a disjoined host region free from the native pathogens of its original home.

The ill informed often wrongly compare such imported plants with the less healthy native cultivated plants, to the latter's disadvantage. Here, as in human experience, the failings of old servants are known and new brooms sweep clean. The imported plants still remain susceptible to their native infectious diseases, only, for the time being, the pathogen is absent. Moreover, as we shall see later, they are also susceptible to new

diseases which spread to them in the new area. The pathogen always follows the host plant, and sooner or later it breaks out in the hitherto clean area. Thus, it is merely the length of the phase interval which has to be considered; how long is it before a pathogen catches up with its host? Three examples may be given.

The potato plant was introduced into Europe from South America during the second half of the sixteenth century and was, at that time, ideally healthy. Its leaves must have remained green and strong until far into the autumn. During the eighteenth century the cultivation of this crop was greatly expanded in central and western Europe, especially in famine years. This was because it provided per unit of field area double the calorific value of cereals although its culinary quality is inferior if the necessary condiments are lacking. The new food habit, of meeting the need for carbohydrates with potatoes rather than with porridge or bread, persisted after the famine years and so, therefore, did the large flourishing healthy potato area.

In the year 1830, potato blight (Fig. 195, *Phytophthora infestans*) was observed in the potato fields of both North America and Europe. In 1835 it invaded Great Britain and from 1840 to 1842 spread generally throughout Europe. The true pandemic attack of potato blight first ran through the European potato area in the damp cold summer of 1845 (p. 138), and thus within a few years brought about the hitherto lacking state of disease saturation. Thus, if potato cultivation be considered as a whole, the time lag between the introduction of the host and the subsequent appearance of the parasite was about 80 years.

As a second example the Douglas fir (*Pseudotsuga Douglasii*) may be cited. In the middle of last century its several varieties were brought to Europe from the western United States, and later it was propagated on a large scale as a substitute for the Weymouth pine which had been decimated by blister rust. It gave rise to the brightest hopes and 'had no enemies'; but these followed later.

The leaf cast of Scottish Douglas fir caused by the Ascomycete *Rhabdochline pseudotsugae* was first noticed in 1911 in the north-western United States, and although it probably reached Scotland before the First World War it appeared there in epidemic form only in 1922; since then it has spread throughout the whole of Europe.

A second needle disease, the leaf cast of Swiss Douglas fir caused by another Ascomycete, *Phaeocryptopus Gäumanni* (Fig. 49), also occurs. It has been known in Ireland, England, and Switzerland since about 1927, has now become pandemic throughout the whole of Europe, and has rendered questionable the cultivation of the Douglas fir in most of its areas.

The dream of a perfect Douglas fir has thus come to an end. The time lag between the introduction of the host and the subsequent appearance of the pathogen was about 60 years.

As a third example, *Puccinia Komarovii* (p. 140) on *Impatiens parviflora*

is instructive because, in this instance, world trade has certainly played no part in the spread of the parasite. The host plant derives from southern Siberia and Mongolia; after 1837 it escaped from the Botanical Gardens at Geneva and Dresden and since then has been growing wild as a garden escape. The rust appeared spontaneously towards the end of the First World War (p. 141) and thus caught up with the host after about 80 years.

Generally, therefore, a period of at least one human generation can be expected to elapse before epidemic pathogens are likely to overtake their hosts. During this period of latency cultivated plants produce their best yields because they are unhindered by disease. The aim of quarantine measures is to prolong this respite as much as possible.

In the case of pathogens with a low dispersal capacity these measures, if applied rigorously, may be permanently successful. Thus, before the First World War, *Citrus* canker (*Pseudomonas citri*) was introduced with diseased plant material from eastern Asia into Texas and the other Gulf states of the United States and grew into a pandemic, especially in Florida. In the absence of any better means of combating the disease, affected trees, worth 12–24 million dollars, were drenched with petrol and burnt. Since bacterioses are not disseminated aerogenously, the epidemic was successfully kept out of California by these means and by strict internal quarantine measures.

In this example, therefore, it was man who first disseminated and later exterminated the pathogen. Were it not for such human (anthropogenic) dissemination by means of infected plant material (p. 91) some diseases would not have entered their new areas until a much later date. The plant pathologist is hesitant, therefore, to introduce even experimentally a great many foreign plants, the 'exotics' of the forester, into new areas of cultivation. Admittedly, two important agricultural crop plants, potato and maize, have been introduced from overseas, but this is no justification for reckless importation. Because of the 'time lag' already discussed the weight of epidemiological argument is frequently ignored and only grandchildren live to see its cogency demonstrated.

In the long run, the only successful way of obtaining heavy-yielding plants both in agriculture and forestry is to choose and systematically breed indigenous and disease-saturated plant material which has been exposed to natural selection for centuries (*Landsorten*).

(cc) A cultivated plant temporarily evades its parasites because resistant varieties are developed.

This is actually the same process as has been described under (bb), with the difference that disease-resistant varieties have been created by artificial breeding from existing cultivated forms.

But here too the pathogen catches up with the host plant. One field under a disease-resistant variety has a selective effect on the whole neighbourhood. The new variety selects from the populations of existing or newly arising biotypes of the pathogen those capable of parasitizing it, i.e.

it acts like a filter. Thus, at first, the disease incidence is low (Table XIV), but it increases from year to year as the congenial biotypes multiply.

TABLE XIV

*The selective influence (filter effect) of a new host variety on a population of *Tilletia tritici* (wheat bunt). (After Roemer et al., 1938)*

Variety of wheat	Attack (%) in the years:					
	1929	1930	1932	1933	1934	1935
Ridit	0.4	14.8	7.4	36.6	32.2	46.6
Heils Dickkopf	—	32.0	37.9	82.2	68.7	91.8
Svalöf's Panzer III . .	57.4	70.0	68.7	93.6	78.9	86.3

'For example, until 1925, race 2 of *Ustilago tritici* (Fig. 98) was found only very rarely in Northern Germany on the spring wheat variety Grüne Dame. But when, in 1926, the variety Peragis, a cross between Grüne Dame and Blaue Dame, was grown in quick succession this race was suddenly presented with the opportunity of multiplying quickly. Race 2 soon became more abundant and its proportion in the air-borne spore material rose from year to year, correspondingly displacing race 1. Previously the latter had thrived on Bordeaux spring wheat, a variety formerly grown on 90% (to-day only 35%) of the area under spring wheat in Germany.

'In Central Germany a large increase of race 7 of *Puccinia glumarum tritici* has occurred over the last 5 years, owing to wider cultivation of the winter wheat, Carsten V. For some years, this variety was free from yellow rust in Holland as initially it had been in Germany. In 1934, yellow rust was found for the first time on Carsten V in Holland, and in 1936 the variety was entirely covered with uredospores. If it were withdrawn from cultivation and then grown again after a break of 5 years, it would remain free of rust for a time until race 7 again became dominant in the population.' (Roemer *et al.*, 1938.)

The probability that some one race of a pathogen will attack a new host variety increases with the number of biotypes which occur in a population of the pathogen in any given area.

This relation holds also between different countries and continents; thus, certain North American varieties of wheat are highly susceptible to some central European biotypes of *Tilletia*. If the latter were introduced into North America, much current breeding work would become obsolete. Hence, it is wrong to argue that since bunt is already present in a given country it can do no further damage because it is the particular biotypes that are decisive. Epidemiologically, a new biotype may have the same importance as a new pathogen.

(*dd*) A cultivated plant in a new area may succumb to a new disease because of the attack of an indigenous parasite.

The cosmopolitan large-scale cultivation of agricultural plants creates a reservoir of diseases from a wide variety of hosts. For instance, it is unlikely that the wild forms of the potato, growing in the Andes, were subject to all the 300 mycoses, bacterioses, and viroses which to-day attack the

cultivated potato the world over. It is much more likely that large-scale cultivation in all parts of the world has attracted pathogens like a magnet, chiefly from the wild Solanaceae, but also from a variety of other hosts. Any pathogen that could attack the potato did so and then spread from continent to continent within the area delimited by the cultivation of the host, thus contributing to the disease reservoir. Hence, new infectious diseases of cultivated plants may arise, quite apart from the development of new pathogens discussed below.

One of the newest of potato maladies, wart disease (Fig. 187, *Synchytrium endobioticum*) almost certainly represents such an increase in the number of diseases. It was first recorded in England and Scotland in the seventies of last century. It is unknown in the country of origin of the potato and hence it is unlikely that it was introduced into Europe with diseased tubers, but must have originated here. The disease may have been present on such weeds as *Solanum nigrum* (black nightshade) or *S. Dulcamara* (bittersweet) without having been noticed. Then in the seventies it began to attack potatoes in neighbouring fields as a 'new disease' in Great Britain.

Although this disease can only be spread by infected seed tubers (p. 111) it extended over the Old and New Worlds during the next decades; 1896 Hungary, 1908 Germany, 1909 Newfoundland, 1918 United States, 1921 South America, &c. Thus, it is really a new disease of potatoes, which was only 'created' by man through the bringing about by him of the meeting of cultivated potatoes and infected weeds, but which has since spread over the entire globe.

(ee) A pathogen undergoes a spontaneous increase of its parasitic faculties and thus becomes the starting-point of a new epidemic.

This aspect of the problem involves biological processes which will be discussed in Section 3 ('Changes in the Parasitic Adaptation of Pathogens'). A new race, i.e. a new pathogen, may arise from a parasitic strain through change of habit, hybridization, saltation, or mutation. The change may be temporary or permanent and may give rise to a new epidemic according to sub-sections (aa) or (bb) considered above. The spontaneous flaring up of endemics believed to be extinct may be due partly to this, but partly also to the cultivation of new host varieties which prove after a time to be susceptible to certain endemic biotypes of the pathogen.

(e) *High Reproductive Capacity of the Pathogen as a Condition for an Epidemic*

Since, by definition, an epidemic is a temporal and local massing of cases of infectious disease, it is clear that only pathogens of high reproductive capacity can give rise to epidemics.

The reproductive capacity of a pathogen is determined by (aa) its fecundity or numerical fertility, (bb) its numerical threshold of infection, and (cc) the rhythm of its successive generations.

(aa) Fecundity of the pathogen (number of disseminative units). Plant

pathogenic bacteria and viruses do not form any specialized disseminative units; the former are transmitted as vegetative cells, the latter by infectious particles. Hence, only in the pathogenic fungi is there any problem of the number of propagative units.

Only fungi of high numerical fertility are able to cause epidemics. *Corticium vagum* (p. 59) which rarely sporulates cannot, therefore, give rise to a progressive epidemic though it can persist by means of its cutinized hyphae wherever it has gained a foothold. The fungi concerned here produce the huge mass of spores needed for an epidemic by the simple and least exacting method of vegetative segmentation (cf. Figs. 73 and 100), i.e. sexual processes such as karyogamy are not involved.

Millardet (cit. after Riehm, 1928) exposed vaselined glass plates in a vineyard which was heavily attacked by downy mildew (*Plasmopara viticola*). After only 26 hours' exposure he counted on the average 32,000 conidia per sq. cm., each of which on germination produces six to eight zoospores.

Sclerospora philippinensis (downy mildew of maize) growing on a medium-sized plant of about 3.5 sq. dm. leaf area, produces approximately 252 million conidia in a single night. On a large plant with a leaf area of 26.3 sq. dm., up to 2,000 million conidia may be produced (Weston, 1923). How many more, therefore, during one complete season!

During one spring season the black rust (*Puccinia graminis*) produces on a medium-sized barberry bush about 64,000 million, i.e. 6.4×10^{10} aecidiospores (Levine, 1928), a figure which is some 30 times the human population of the world. Under favourable conditions these spores can give rise to uredosori on cereal plants within 10 days. If each aecidiospore produces only one uredosorus, and if a uredosorus cuts off 200,000 or 2×10^5 uredospores we have, after 10 days, 1.28×10^{16} uredospores. Until the end of July a further six generations may ensue which brings the uredospore count up to 10^{46} . This figure is astronomical; according to O. Thomas in *Astronomy*, the ratio of the radius of the universe to that of the proton is 'only' 10^{40} . The mass of Pfeiffer's influenza bacillus is 10^{-15} g., that of the earth 10^{28} g., hence the earth is 'only' 10^{43} times the weight of the influenza bacillus. To think that the figure for the offspring from a single barberry bush amounts to 10^{46} !

Although, of course, many links in this 'snowball' chain may be broken, this enormous progression explains why in cereal-growing countries the whole air space becomes heavily charged with rust spores to a height of 4,000 m. (p. 120). The probability of a host plant being hit thus reaches such dimensions that every plant in the whole area of cultivation is certain to be infected more than once.

(bb) Numerical threshold of infection. Pathogens with a low numerical threshold of infection (p. 38) have, because of this, greater reproductive capacity than those with a higher threshold. If one smut spore of *Ustilago tritici* (p. 108) is carried 500 m. by the wind, alights on a wheat stigma, and germinates there, the end result is epidemiologically unimportant, since it

cannot by itself give rise to infection. By contrast, even single spore infections with aecidiospores of *Cronartium ribicola* (p. 145) on *Ribes* leaves and *Puccinia graminis* (p. 84) on cereal leaves can be effective.

(cc) The rhythm of successive generations (length of life cycle). In spite of the numerical fertility mentioned above, three to four generations are required by *Plasmopara viticola* (downy mildew of vine) to start an epidemic (Zillig, 1942). Similar relations may obtain in all other infectious diseases

of plants. However, since in temperate climates the vegetative season is limited and, moreover, since only part of this is available to the parasites, generally at most 5 months, the speed with which generations succeed one another is one of the decisive criteria of the epidemiological capacity of a pathogen.

The rate of succession of generations in any pathogen is determined by the time required for the production of fructifications and the formation and germination of propagative spores.

The time required to produce reproductive bodies (p. 37). The loose smut of wheat (p. 108) and the cluster cup rust of pear trees (p. 86) are two examples of long-delayed fructification; in the former the period is one year and in the latter, on the juniper, even several years. Both, therefore, are ill adapted to cause progressive

epidemics; they result only in a local, stationary, or non-progressive accumulation of diseased individuals.

Epidemiologically species of powdery mildew are very efficient, their time to fructification being 6–8 days, as are also potato blight, downy mildew of vine, and cereal rusts, in which the time to fructification is about 10 days, &c. In these fungi several generations are passed through in a single month with a corresponding progressive building up of spore numbers.

The speed of formation of propagative spores. Since propagative spores, in contrast to resting spores, are produced asexually, i.e. without the complex processes of fertilization and karyogamy, they can arise within a few hours, provided the temperature is favourable and the moisture content of the air is high. It is a popular belief that downy mildew arises 'overnight', a statement expressing two facts: that light normally inhibits the formation of asexual fructifications, and that the few hours of a warm summer night are sufficient for the formation of a mat of conidiophores.

The speed of germination of propagative spores. Here two factors come into play. Epidemics are possible only with those fungi (1) in which at

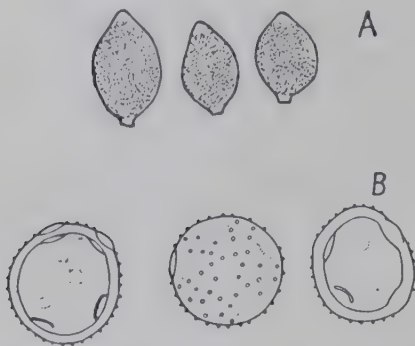


FIG. 127. Propagative fungal spores about to germinate. A conidia (zoosporangia) of *Phytophthora infestans* (potato blight) with apical germ papillae. B uredospores of *Uromyces airae flexuosae* (rust of grasses), the exospore sculptured with fine warts and showing germ pores probably filled with a pectin-like substance which causes them to bulge outwards, and often also inwards, like papillae. A $\times 330$, B $\times 440$. (After Krüger, 1939, and Guyot, 1938.)

least one spore form can germinate immediately, i.e. without a rest period intervening; and (2) whose spore germination is completed within a few hours. The fulfilment of these conditions is provided for by means of thin walls or occasionally by the formation of definite points of germination. Thus, a terminal germination papilla is found in the conidia of the potato blight



FIG. 128. Turnips (*Brassica Rapa*) showing club root tumours (*Plasmodiophora brassicae*). Approx. $\times \frac{1}{4}$. Original at the Federal Research Institute, Wädenswil.

fungus (Fig. 127, *A*) and special germination pores in the uredospores of the rust fungi (Fig. 127, *B*).

(*f*) *Ready Dissemination of the Pathogen as a Condition for an Epidemic*

The epidemic potential of a pathogen is determined not only by the number of its propagative units, but also by the facility with which they are dispersed. In the epidemiogenic mycoses transmission is effected mostly by the wind; in bacterioses and viroses by insects. Here the epidemic capacity of the mycoses only will be considered, since only fungi give rise to special propagative units of dissemination.

The latter, conidia, oidia, uredospores, &c., must be physically capable of being transported by the wind and biologically able to survive it.

Physically, the first condition is the opportunity to get into the air-stream so as to become readily wind-borne. Hence, only those plant pathogenic fungi can cause epidemics which form their propagative spores

on the external surface of the host (Fig. 100) or eject their spores into the open in some other way, e.g. pycnidia. Fungi which reproduce inside the host tissue, even if their spore production be vast as, for instance, in club root (Fig. 128) and maize smut (Fig. 144), can initiate only circumscribed and not true progressive epidemics.

The second condition for air-borne dispersal, a light weight, is satisfied by all fungal spores because of their small dimensions (p. 116).

As regards the biological fitness of propagative spores, i.e. their ability to survive air-borne transport with unimpaired capacity to germinate and infect, the position is less favourable. As was mentioned above, fungal propagative spores are usually formed rapidly with little expenditure of materials and are often surrounded by only a thin outer wall. Their length of life and resistance to environmental factors are correspondingly low, at least in comparison with resting spores (p. 104): they are ephemeral in character.

Nevertheless, conidia and oidia, to deal with these first, retain their power to germinate for at least a few days, and under optimal conditions for several months. But the time needed for germination to occur often increases with age (Table XV), and the spores may easily be too late, or be able to make only limited use of favourable environmental conditions.

TABLE XV

The influence of age on the germinability and speed of germination of the conidia of Diplocarpon rosae (black spot of rose). (After Frick, 1943)

<i>Age of spore</i>	<i>Germination</i>	<i>Onset of germination</i>
(days)	(%)	(hours)
3	92	14
6	88	14
9	71	24
14	66	36
19	45	48
23	17	60
27	6	72
31	0	—

Their greatest danger is from the dryness of the air in which they float. Thus, the length of life of the conidia of downy mildew of vine and of potato blight (*Plasmopara viticola* and *Phytophthora infestans*) is only a few days in fine summer weather. On the other hand, conidia of potato blight may retain their germinability for 2 months in moist soil, or for over 2 years in a peat soil (de Bruijn, 1922). It is, however, not known whether they are then still able to cause infection.

Under natural conditions the oidia of the powdery mildews of wheat and barley (*Erysiphe graminis tritici* and *E. g. hordei*) lose their power to infect after only 48 hours (Metzger, 1942). Under laboratory conditions, however, with high atmospheric humidity (95%) and low temperature (near 0° C. to prevent germination), wheat mildew may retain it for 70 days and

barley mildew for 51 days. Sometimes single oidia have considerably thicker walls than usual, and one is inclined to assume that they are therefore more durable, rather of the nature of gemmae.

There are, of course, exceptions to the general rule that conidia and oidia remain viable for only a short period: for instance, conidia of the ergot of rye (*Claviceps purpurea*) show an astonishing viability; they can even overwinter successfully in the small apical 'caps' of the sclerotia (Fig. 80; Stäger, 1912).

In spite of their similar propagative character uredospores, e.g. of cereal rusts, generally survive longer than conidia and oidia. Those of brown rust of wheat (*Puccinia triticina*), brown rust of rye (*P. dispersa*), and crown rust of oats (*P. lolii avenae*), for instance, remain unharmed after 4 hours in direct sunlight, but those of yellow rust of wheat (*P. glumarum tritici*) are injured (Gassner and Straib, 1928).

As regards viability, the optimum relative humidity of the air is about 40-60%, i.e. approximately the air humidity in Atlantic climates on fine summer days. Uredospores of yellow rust (*P. glumarum*) retain their ability to germinate for 433 days at 40% relative humidity and a temperature of 0° C., for 179 days at 5° C., and for about 50 days at 15° C. (Becker, 1928). Thus the length of time for which they remain viable decreases quickly with rising temperatures. The same is true for their infective capacity (Table XVI). Since low temperatures prevail in higher regions of the atmosphere they are, in these layers, able to remain infective for the whole of the vegetative season. Hence the great danger of air-borne transport of cereal rusts (p. 117) in the upper strata of the atmosphere.

TABLE XVI

The approximate duration of the infective capacity of uredospores of black rust of wheat (Puccinia graminis tritici) biotype 3, at various temperatures and relative air humidities. (After Peltier, 1922)

Approximate relative air humidity	Temperature of storage (° C.)				
	5°	10°	15°	20°	25
(%)	(days)	(days)	(days)	(days)	(days)
90	7	< 7	< 7	< 7	< 7
81	14	14	7	7	< 7
70	112	112	14	7	< 7
61	112	98	98	< 7	14
49	112	112	105	7	14
38	105	98	98	7	< 7
30	28	21	7	7	< 7
22	28	14	7	< 7	< 7
11	< 7	< 7	7	< 7	< 7

Finally, the example of apple scab (*Endostigme inaequalis*) will show to what extent the length of life and the general hardiness of propagative spores are factors limiting their epidemiological fitness (Herbst, 1941). The ascospores are resistant to environmental conditions, and after

ejection (pp. 116 and 164) may be carried to any distance, and continue the infection chain.

Conidia behave differently. Their transportation by the wind for miles serves no purpose, because they are so sensitive to dry and warm conditions that en route they lose their ability to germinate. Their only chance of dissemination is carriage in small raindrops blown by the wind. Hence, their spread is limited to a circle of about 10–20 m. radius round the parent tree (p. 113).

Physically, the conditions for dispersal by the wind are the same for both types of spore, yet the ascospores alone can cause a progressive epidemic, whereas the conidia can start merely short-range infections.

(g) *Unexacting (Euryzoic) Growth Requirements of the Pathogen as a Condition for an Epidemic*

Weeds never die out, because they are able to adapt themselves to every situation: epidemiogenic pathogens are such weeds. They can produce epidemics because they are not selective and can support a wide range of variation in each of the eight conditioning factor groups outlined above (p. 140), i.e. they are easily satisfied or euryzoic (p. 26) in their ecological demands.

Fortunately, such a many-sided adaptability as is necessary for the initiation of epidemics is not often realized among plant pathogens; therefore relatively few plant pathogens can give rise to epidemics, and hence their host plants continue to survive.

For example, the tinder fungus (*Polyporus fomentarius*) causes a lethal white rot of beech trees. Its fecundity is enormous, one fructification producing in one vegetative season approximately 9–18 billion, i.e. $9-18 \times 10^{12}$ basidiospores (Buchwald, 1938) and this may be repeated annually for 50–100 years. Thus, judging by the number of spores shed, this disease should run epidemically through our beech woods, yet it has become rare in central Europe. It would seem that its spores, besides quickly losing their power of germination, must make very special and narrowly defined (stenozyotic) demands on the environment for their development. They appear to have a high numerical threshold of infection, and their requirements for successful attack are so infrequently realized that the mere cutting down of diseased trees by the forester has been enough to cause the malady to fade away. In the case of the potato (*Phytophthora*) blight with its low infection threshold, such measures would not be successful.

(h) *Optimal Weather Conditions for the Development of the Pathogen as a Condition for an Epidemic (Meteoropathology)*

Meteoropathology is the study of the relation between weather conditions and epidemics. In human pathology it is based chiefly on data from meteorological stations. In plant pathology these need to be supplemented by microclimatic determinations of the air close to the soil because, in many cases, even the nocturnal formation of dew is sufficient to permit infection if the air temperature be high enough. For this reason, also,

particular local conditions may play a decisive role, e.g. downward currents of cool air during the night near the mouths of lateral valleys or strong radiation at night above areas of dark peat, &c.

The critical point at which the course of an epidemic is influenced by the weather differs in human and plant diseases.

The medical aspect of meteoropathology is mainly concerned with the influence of the weather on the disposition of the host prior to infection. For instance, in the case of the annual spring epidemics of influenza and measles, it may be assumed that the pathogens are present everywhere, i.e. that they are ubiquitous. Often, sick individuals have already been infected for a long time without showing any symptoms (p. 5) before the disease becomes manifest. The outbreak of the epidemic generally depends on a heightened disease proneness of the host, here determined by seasonal malnutrition (cyclic avitaminosis) of the whole population and by the weather.

The same holds true for the further course of the disease after infection has taken place. In the human body the pathogen is maintained under uniform conditions of temperature and moisture and is thus protected from any direct influence of the weather, which can only affect the host. For example, the severe neurotropic form of poliomyelitis (infantile paralysis) shows a marked increase during midsummer, which is apparently determined by a climatically induced alteration in the resistance of infected individuals.

In plant pathology the circumstances are different. Weather can cause no important alteration in the disease proneness of plants, as we shall see in Chapter 4, because the effects of severe conditions in winter and early spring are normally excluded, vegetation during this period being dormant.

Moreover, only in very rare cases can it be assumed that the plant pathogen is ubiquitous (p. 155), and even then only at the height of the epidemic. Normally the disease spreads radially in a series of infections from a primary focus. This needs to be in an optimal environment to allow of the abundant formation of reproductive bodies at the surface of the host and of their subsequent dispersal. The new infections, again being favoured by optimal conditions, must take firm hold, and in turn form reproductive bodies which are dispersed and give rise to further infections, &c., until after a few generations epidemic proportions have been reached. The emphasis of discussion in meteoropathology in the botanical realm falls, therefore, on the favourable effect of weather on the pathogen and not on its influence on the disposition of the host to disease. Especially important are its effects on reproduction, e.g. on the rate of spore formation or fructification and the speed of succession of generations or incubation, and on infection itself, in particular on spore germination. Not only the plants themselves but also their parasites live much more in the open than does man. Consequently the climatic factors of air temperature and atmospheric moisture, including precipitation with its diurnal and seasonal distribution, play the main role in plant epidemiology.

The optimal epidemiological conditions for reproduction and infection may be alike or not, according to the particular pathogen concerned.

In the vast majority of cases they are identical, because both reproduction and infection are favoured by high atmospheric humidity, such as evening rain, mists that wet leaves on both sides, and dew. The downy mildew of vine (*Plasmopara viticola*, p. 103) is a good example.

The mycelium inside the leaf stops growing at temperatures below 10–12° C. and above 30° C., but it is not damaged by exposure to frost nor



FIG. 129. Young grape covered with a mat of conidiophores of *Plasmopara viticola*. Approx. $\times 12$. (After Müller and Sleumer, 1934.)

to temperatures as high as 43° C. and it resumes growth on the return of favourable temperatures. The optimum air temperature for fecundity and speed of reproduction lies at about 22–24° C. (Fig. 130). After only 4 days at this temperature a mat of conidiophores (Fig. 129) appears on the leaves and first buds of the vine, protruding through the stomata. But this takes place only during darkness, i.e. between 1 and 3 a.m. With an air temperature of 15° C. the pre-fructification period is lengthened to 8 days and the speed of advance of the infection chain is halved.

The development and abstriction of conidia are also favoured by high air temperature and humidity (100% dew formation) during the night. If the relative humidity is only 80–90% the pre-fructification period is increased by

2–4 days, *ceteris paribus*. Below 60% relative humidity the pathogen does not sporulate at all, the infections remain sterile, and the infection chain is broken.

The same ecological factors that favour sporulation of the downy mildew also favour the germination of its conidia and the success of infection. At optimal temperatures, 18–24° C. (Fig. 31), germination may take place in 1–2 hours. Thus, if the conidiophores which have emerged through the stomata complete the abstriction of their conidia by 2 a.m., germination and infection may be accomplished successfully even before daybreak. Four days later, under the same external conditions, another link will have been added to the infection chain: in this way a local infection focus may expand into an epidemic within 3 weeks.

The most important meteorological conditions for a *Plasmopara* epidemic are, therefore, warm and humid nights with heavy dew formation, or mist or general or evening rain. Their effects are enhanced if the number of hours of sunshine be limited, for then foliage and soil tend to dry more slowly.

Using these facts of meteorology, the Phenological Plant Protection Service tries to forecast epidemic outbreaks of *Plasmopara*. It determines when the climatic conditions are optimal for the sporulation of the pathogen and for the infection of new hosts, i.e. when an epidemic is likely to break out and when the full chemical protective measures should be employed; i.e. it formulates a 'spray calendar'.

Predictions of this kind, like the weather forecasts of meteorological stations, are generally a thankless task. But short-term and local prognoses

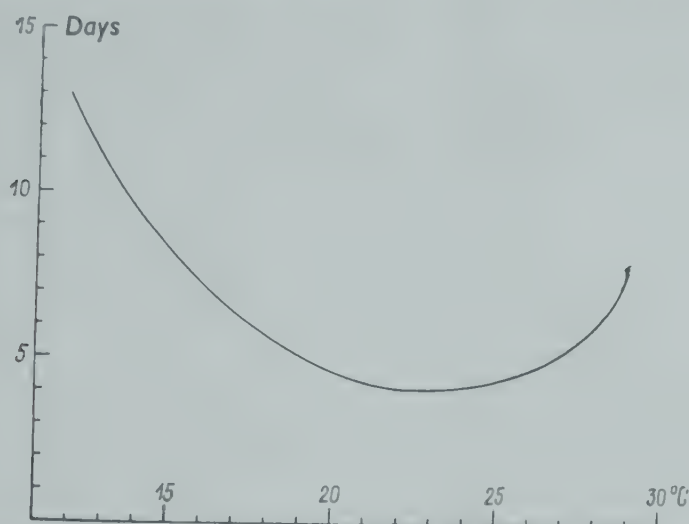


FIG. 130. The influence of air temperature on the time to sporulation of *Plasmopara viticola* (downy mildew of vine). (After Müller and Sleumer, 1934.)

may reach a sufficient degree of likelihood to have practical value, especially in areas of low precipitation, where they are useful above all at the time of transition from primary to secondary infection.

TABLE XVII

Average meteorological air temperature and the estimated and actual times to sporulation of Plasmopara viticola in a given area. (After Müller and Sleumer, 1934)

Period of time	Air temperature	Estimated time to sporulation	Actual time to sporulation
	(° C.)	(days)	(days)
11-20 May . .	13-14	10	15-18
21-31 May . .	15-15.5	8	12-15
1-10 June . .	17-17.2	6-7	10-13
11-20 June . .	16.2-16.5	7-8	8-10
21-30 June . .	15.7-18.5	6-7	6-7
July and August. .	18-19.5	5-6	5-6

In the first two columns of Table XVII are shown the mean air temperatures determined at Karlsruhe over a period of many years. In column 3 the estimated times to sporulation for different mean temperatures are presented, as calculated from the data shown in Fig. 130. Finally, the actual times to sporulation, based on field observations, are given in

column 4. These two values show good agreement from mid-June to the end of August. Hence, over this period, the time to sporulation can be predicted with sufficient accuracy from the average temperatures. Before mid-June, however, the actual times are much longer than those predicted meteorologically, for then cool nights with temperatures below 12°C . tend to stop the growth of the mycelium. After each check there is a lag-period in growth which leads to a corresponding lengthening in the time to sporulation.

Table XVII, therefore, provides a basis for the prediction of *Plasmopara* epidemics in the Karlsruhe area. With the help of this Table a grower can

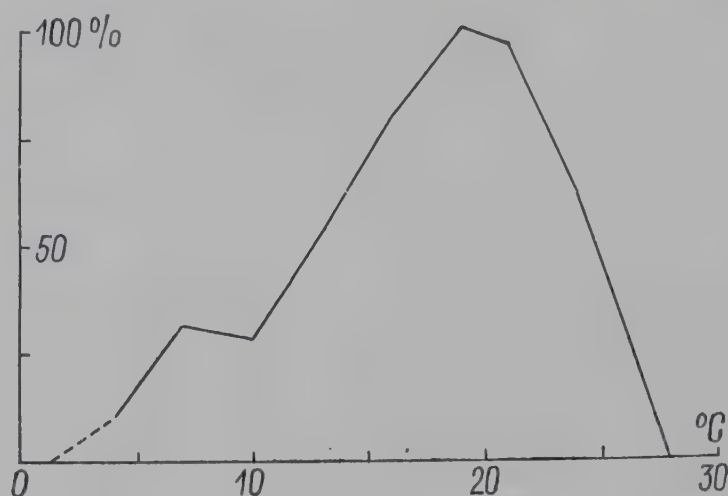


FIG. 131. The influence of air temperature on the rate of maturation of the ascospores of *Endostigme inaequalis* (apple scab). Abscissae: air temperature. Ordinates: % asci containing mature ascospores in apple leaves collected out of doors. (After Wilson, 1928.)

estimate the interval after successful infection at which a new *Plasmopara* generation will be formed, provided the weather is damp. To break the infection chain the vineyard must be sprayed prophylactically during this interval so that the new wave of infection is prevented by killing the conidiophores when they first appear.

For most other mycoses similar external conditions are necessary as for epidemics of *Plasmopara*.

Optimal air temperatures during the winter (Fig. 131) accelerate the development and maturation of infection foci and thus the start of primary infections. Sporulation is also more abundant and the time elapsing before it occurs is reduced (cf. Table XVIII). For this reason, waves of infection follow one another at ever shorter intervals in early summer when temperatures are rising.

The optimum moisture content of the air for spore formation in nearly all other fungal pathogens lies similarly at about 100% relative humidity. Epidemiologically, rainfall has two opposing effects: on the one hand it brings down the propagative fungal spores floating in the air, and on the other hand, it creates optimal conditions for the formation of new spores.

Light generally has the same effect as on *Plasmopara viticola*. It is less

important than air temperature and atmospheric humidity; it appears slightly to inhibit the formation of propagative spores but seems to favour their further development and maturation. *Fusarium bulbigenum* var. *batatas* Woll. (wilt disease of sweet potato), produces mainly 4- to 5-celled conidia when growing in the light, whereas those produced in the dark have only 2 cells (Harter, 1941). The length of the 3-celled conidia of *Fusarium caeruleum* (potato dry rot) is also affected by the amount of light: cultured in full sunlight they are $42.2 \pm 0.23 \mu$ in length; if exposed to daylight from 9 to 12 a.m. and kept in darkness for the rest of the day they are $35.5 \pm 0.26 \mu$; and if maintained continually in the dark they are $29.5 \pm 0.17 \mu$ (Harter, 1939). Changes in septation and size of the conidia, therefore, induced by different light conditions much exceed the morphological limits of varietal difference. Hence it is possible that, under certain circumstances, cultures maintained permanently in the dark, e.g. in incubators, instead of under the natural alternation of light and darkness, may also show changes in their aggressiveness. The behaviour of ergot of rye points in this direction since it produces more pigments and ergosterol in sunlight than in the dark (McCrea, 1931). It is permissible, therefore, to suppose that daylight may induce changes in vital substances not only in warm-blooded animals, such, for example, as the activation of provitamin D, but perhaps also changes in certain phytopathogenic micro-organisms.

TABLE XVIII

The influence of air temperature on the average time taken by three species of rust fungi to produce their first uredosori. (After Gassner and Appel, 1927)

Species of rust	Air temperature (° C.)		
	10°	15°	20°
	(days)	(days)	(days)
<i>Puccinia dispersa</i> (brown rust of rye)	15.3	9.2	6.7
<i>Puccinia triticina</i> (brown rust of wheat)	19.0	11.7	8.0
<i>Puccinia lolii</i> (crown rust of wheat)	19.0	11.1	7.8

So far as is known, spore shedding occurs chiefly by night, as in *Plasmodiophora* (before midnight in the example shown in Fig. 132).

The powdery mildews (Erysiphaceae) afford an unusual example in that the reproduction of the pathogen and the infection of the new host show diverging ecological requirements. Here, both the number of oidial spores released and their germinability are greater if they have developed in bright sunlight than in rainy or dull weather (Tables XIX and XX).

The daily cycle of spore formation, or sporulation, thus takes a course different from that shown in Fig. 132. Their spore mother cells, like those of other fungi, begin to cut off new daughter cells in the early afternoon, and the number of cells in the oidial chains increases towards the next morning (Fig. 133). But the abstriction, rounding off, and eventual shedding of the single oidia take place during the day, preferably during dry

weather and in direct sunlight. Hence, in Fig. 133 the number of cells in the oidial chains falls steeply during the morning. The actual cutting off is retarded in the dark, so that the number of oidia in each chain is greater in the absence than in the presence of light (Table XIX).

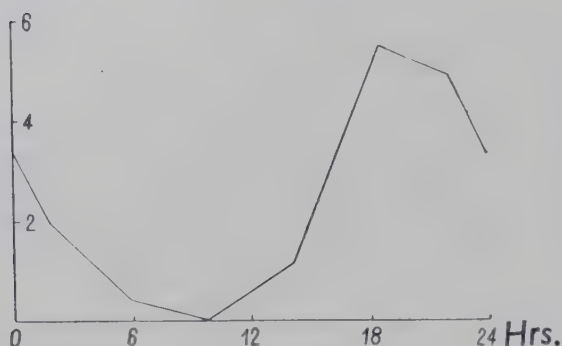


FIG. 132.

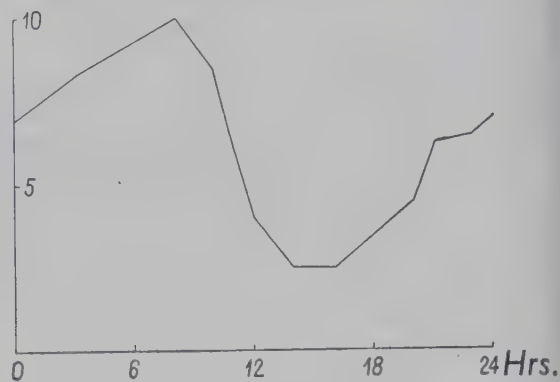


FIG. 133.

FIG. 132. Daily rhythm of ascospore ejection in *Taphrina deformans* (peach leaf curl). Abscissae: time of day. Ordinates: mean number of ascospore groups caught every hour on a slide 34 sq. mm. in area. (After Yarwood, 1941.)

FIG. 133. Daily rhythm of cell numbers in the oidial chains of *Sphaerotheca pannosa* (powdery mildew of rose). Abscissae: time of day. Ordinates: mean number of cells in the oidial chains. (Diagrammatic after Childs, 1940.)

On account of this favouring effect of direct sunlight on spore dispersal, espalier roses on sunny walls of houses are more severely attacked by mildew than are roses grown in the open. Similarly, stool shoots of oak on the sunny edges of woods are attacked more heavily by oak mildew than those in the middle of the stand.

However, as usual, oidia require liquid water or at least heavy dew for their germination.

TABLE XIX

The influence of light on the vitality of certain Erysiphaceae.
(After Hammarlund, 1925)

Species of fungus	Host plant	Darkness		Diffuse daylight		Sunlight	
		No. of oidia per chain	Germination	No. of oidia per chain	Germination	No. of oidia per chain	Germination
<i>Sphaerotheca pannosa</i>	<i>Rosa</i> sp.	8.7	(%) 0	6.5	(%) 50.4	6.0	(%) 66.3
<i>Podosphaera leucotricha</i>	<i>Pyrus malus</i>	9.0	0	6.7	76.6	6.2	86.9
<i>Erysiphe graminis</i>	<i>Avena sativa</i>	9.3	3.7	8.2	38.7	8.1	45.8

An epidemic of powdery mildew thus demands the coincidence of two opposing meteorological factors. On the one hand, dry weather, high temperature, and bright illumination are necessary for spore formation, and on the other hand, liquid water is needed for germination of the spores.

But in midsummer these two seemingly opposed conditions are met by the simple alternation of day and night; clear warm days favour vigorous production of oidia, while equally clear nights with strong radiation and dew formation allow the oidia to germinate.

TABLE XX

The influence of air temperature and air humidity on the fecundity and the vitality of the oidia of Sphaerotheca pannosa (rose mildew) on Rosa sp. (After Hammarlund, 1925)

Air temperature during development (° C.)	No. of oidia per chain		Germination (%) at 18° C.	
	Dry air	Moist air	Dry air	Moist air
5	4.3	—	9.1	—
10	9.3	4.8	27.6	3.6
15	13.7	—	58.5	—
20	14.6	7.3	89.6	14.2
25	15.9	8.4	84.3	39.8
30	15.0	—	91.6	31.8
35	11.8	5.1	45.3	9.5

(i) Cause of the Progressive Severity of Epidemics

Why do the momentum and the persistence of annual and secular epidemics increase at first both quantitatively, in the number of diseased individuals, and qualitatively, in the severity of the disease? Two causes may be responsible for this.

(aa) Progressive spore dissemination. It has been pointed out above (p. 161) that plant epidemics normally spread radially from local infection foci in accordance with the principle of successive infections. Only rarely is the pathogen ubiquitous and the actual epidemic started by an increase in the disease proneness of the host.

In every epidemic starting from a local infection focus the numbers of germs in the successive links of the chain increase in geometrical progression. The cycle starts with a few germs only, scarcely exceeding the numerical threshold of infection; this is followed by increased germ dissemination → an increased number of diseased host individuals → renewed increase of spore dissemination, &c., in the way described for black rust (p. 155). The epidemic potential of the pathogen thus rises correspondingly.

At the height of the epidemic, progressive spore dissemination has attained such proportions that every individual host plant in the area is not only reached by the pathogen, but infected by a massive dose far exceeding any threshold. Thus, the dispersal density becomes so great that the infection doses attain mass proportions and the probability of infection rises to 100%.

This increase in the density of dissemination and the amount of infective material has quantitative as well as qualitative consequences.

Quantitative effects. As the infection becomes continually denser and

heavier, even the relatively more resistant individuals which at first were able to withstand the onslaught eventually succumb, thus giving the epidemic its all-inclusive character. Finally, then, all individuals within the area, whether susceptible or partially resistant, become its victims.

Qualitative effects. As the amount of spore material grows, it becomes increasingly easy for the pathogen to start infections; hence, the incidence and the severity of the disease increase together to a maximum. Rising numbers of affected individuals and growing severity of the disease are thus the essential attributes of the expanding phase of an epidemic.

(*bb*) Improvement in the parasitic adaptation of the pathogen. In addition to the numerical increase of the pathogen, which causes infection to become continually denser and more massive, a qualitative improvement may also occur in the pathogen during the ascending phase of the epidemic. Here, two distinct effects coincide: (1) increase in the aggressiveness and pathogenicity of individual parasite strains, and (2) increase in the aggressiveness and pathogenicity of parasite populations.

1. Increases in virulence of individual parasite strains. It will be shown in the next chapter, which discusses the problem of passage, that the virulence of certain parasites is increased by passage through congenial hosts. Thus, the parasitic efficiency of the clones increases during the first few generations of an epidemic, so that their parasitic adaptation improves simultaneously with progressive spore dissemination, thus reinforcing the latter process.

2. Increases in virulence of parasite populations. Epidemics are usually started by swarms of different strains, populations of biotypes (pathogenic races). During the epidemic these populations may be selected by the host or by competition between the pathogenic strains themselves, in such a way that the more active biotypes become dominant.

For instance, selective elimination of biotypes by the host occurs in potato blight (*Phytophthora infestans*). At the start of an epidemic highly susceptible potato plants are available which permit stronger and weaker biotypes to grow equally abundantly. Perhaps all strains gain in vitality through these favourable passages, whereby the average virulence of the biotype mixture reaches an optimum value. However, after the most susceptible plants have been eliminated it is the turn of the more resistant ones to be attacked. The less aggressive biotypes cannot develop sufficiently well on these; their vitality decreases until they can no longer exist, and eventually they disappear. Thus, the population undergoes selection in favour of the more efficient strains. Their virulence need not necessarily be higher than at the start of the epidemic, but as the less aggressive strains have been eliminated through selection by the host, the average virulence of the surviving population is higher than that of the initial mixture. Hence the epidemic increases in severity towards its climax.

In this case the shift in virulence affects only the parasitic population, not the individual biotypes; it is based on a filtering of the swarm of parasites by the host resulting in selection in a positive direction.

Selection of biotypes through mutual competition occurs, for instance, in the case of *Bacterium Stewartii* which causes wilt of maize in North America. Here, however, the conditions are very exceptional being directly opposite to those in the case of *Phytophthora* because susceptible maize plants tend to weaken the parasite and resistant ones to stimulate it; hence the effects on selection are reversed in susceptible and resistant maize plants.

If a bacterial suspension is prepared consisting of 90% of a strongly pathogenic strain and 10% of a weakly pathogenic strain, both recognizable

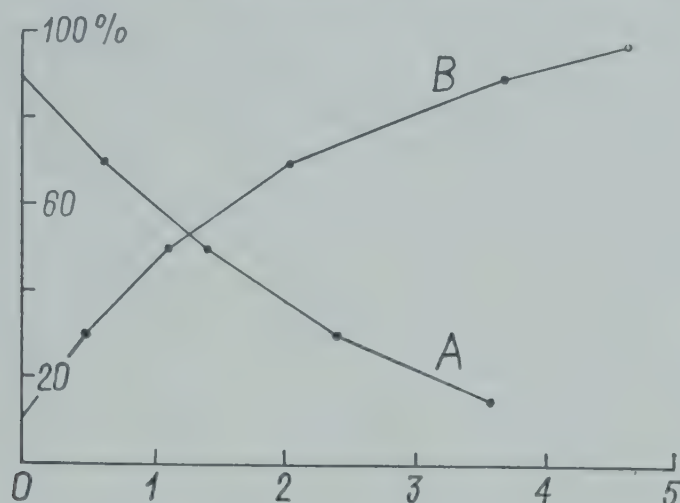


FIG. 134. Influence of the host on the percentage composition of a mixed infection of *Bacterium Stewartii*. Abscissae: duration in months. Ordinates: proportion of strongly or weakly pathogenic bacteria in the mixture. Explanation in text. (After Lincoln, 1940.)

morphologically, and if this suspension be inoculated into plants of a relatively susceptible variety of maize, the mixture of pathogens being transferred repeatedly from diseased individuals to new individuals of the same variety, then the proportion of the strongly pathogenic strain falls from 90% to 15% after four such passages (Fig. 134, curve A). The strongly pathogenic strain has thus been replaced by less pathogenic and more saprophytic individuals. This process is unimportant during the ascending phase of the epidemic curve because, under natural conditions, the strongly aggressive strain will have killed all susceptible host individuals by this time.

More important to our immediate problem is the opposite experiment in which a resistant maize variety is inoculated with a mixture of 10% strongly and 90% weakly pathogenic bacteria. After five passages only 2% of the mixture consists of the weak strain (Fig. 134, curve B), which has thus been swamped almost completely by the strongly pathogenic strain.

When, therefore, during the course of the epidemic, the susceptible maize plants are killed, the pathogenic population undergoes selection on the remaining resistant host individuals. As in the example of *Phytophthora* the direction of the process is positive but its basis is different, being here the mutual competition between pathogenic strains. The dominance

of the more efficient pathogenic strains in this instance also causes an increase in the severity of the epidemic towards its climax.

If at the start of an epidemic the several groups of factors coincide, i.e. progression of spore dissemination, a true rise in the virulence of individual strains of the pathogen, and also an increase in the average virulence of the pathogenic population due to selection, then the epidemic is severe from the beginning.

On the other hand, the complexity of all these factors makes it difficult to give a prognosis of the course of an epidemic which has just begun.

3. *The Conditions for the Decline of an Epidemic*

Why does an epidemic tend to subside, to assume a milder character, and to confine itself within narrower limits just when the spread of infection is at its highest?

In the case of annual epidemics climatic factors may be chiefly responsible; for instance, in the late summer a few cool nights may be sufficient to slow down or arrest the further progression of the pathogen.

In the recession of secular epidemics at least three groups of determinants seem to be involved: (a) an increasing saturation of the host population by disease; (b) a reduction in the disease proneness of the host plants; and (c) a decrease in the vitality of the pathogen.

(a) An Increasing Saturation of the Host Population by Disease as a Condition for the Decline of an Epidemic

When a wild or cultivated variety of plant grows in the absence of a given pathogen, natural selection of resistant individuals by the parasite cannot occur. Possibly, the susceptible individuals may even become dominant or be cultivated preferentially because of their ready growth. For example, before 1830 it was impossible in Europe to select potato stocks for their resistance to *Phytophthora* and it is not improbable that actually the more susceptible, leafy varieties were preferred because of their greater yielding capacity.

Progressive epidemics make good this lack of selection in relation to given pathogens; in the 'ideal' case elimination by them of all susceptible or semi-resistant host individuals is complete, and only truly resistant host populations are left behind: the plant stock has been saturated by the progressive epidemic and become immune to it.

The expression 'disease saturation' has, therefore, a narrower meaning botanically than in either human or veterinary medicine. Endemic 'children's' diseases such as measles, scarlet fever, diphtheria, &c., have saturated human adults (1) by eliminating highly susceptible individuals at an early age, and (2) by inducing an acquired immunity lasting for life. Emphasis is laid on the second aspect, the acquisition of immunity.

In plant diseases, however, acquired immunity has practical importance only in special cases, as will be shown in Chapter 4. Here, therefore,

emphasis is laid on the first aspect, on selection, the elimination of all minus variants, and the destruction of all susceptible individuals.

The extent of disease saturation tends to differ in wild and cultivated plants.

In wild plants, as a rule, natural selection is far-reaching because it operates uninterruptedly over long geological periods. Susceptible individuals become diseased when young, are suppressed by their competitors, prevented from reproducing, and do not reappear; the genetic stock itself is eliminated. Thus species which in and since Tertiary times have developed together with their pathogens have generally reached an equilibrium with them; the pathogen and the disease have become endemic, they still persist in a mild form but no longer affect the vital activities of the surviving selected hosts. Hence, they appear to be innocuous; e.g. *Endothia* in the Mediterranean region (see p. 150).

In cultivated plants, however, rigorous selection by the pathogen is seldom fully accomplished because of human interference. Thus in well-protected seed-producing establishments man raises valuable plant stock which is high yielding but usually more or less susceptible and which needs to be replaced from time to time during further cultivation by fresh seed material. By this replenishment of the stock from an artificial source of cultivation, the cultivated plant is withdrawn from the operation of natural selection.

To take an extreme example. A wild plant species highly susceptible to damping-off or any similar seedling disease will be eliminated in the seedling stage and disappear. On the other hand, a sugar beet variety, however susceptible it may be to damping-off, is protected from the free play of natural forces by cultivation methods and, moreover, is multiplied artificially in seed-growing establishments.

Another reason why disease saturation of cultivated plants is scarcely likely to reach the same high level as in the wild flora is to be found in chemical methods of plant protection. As members of the third generation we may ask ourselves whether it was not fortunate for viticulture that at the time of the original epidemic there was no means of fighting the powdery mildew of vine (*Uncinula necator*, p. 138). At that time, with enormous losses to the people then living, the whole stock of vines was spontaneously saturated by the disease and thereby 'cleaned', so that nowadays the disease is relatively harmless. The downy mildew (*Plasmopara viticola*), however, could at once be fought by spraying with Bordeaux mixture, &c.; hence, the natural saturation of vines has not taken place, and to-day our vines are probably as susceptible to *Plasmopara* as they were 50 years ago.

(b) *A Reduction in the Disease Proneness of the Host Plants as a Condition for the Decline of an Epidemic*

The disease proneness of a host lessens when its disposition abates or when it becomes immune. Both factors are important in human epidemiology.

Cyclic epidemics of man are often caused by a temporary increase in disposition. Thus, if cyclic avitaminosis (p. 143) be eliminated, the influenza epidemics of early spring tend to subside, in spite of the omnipresence of the pathogen. On the other hand, many epidemic diseases, e.g. children's diseases, induce permanent immunity which protects the host from a second attack of the disease. When such an epidemic breaks out in a new region (e.g. The Faeroes or the South Sea Islands), the epidemic eventually subsides to a low level with the increase in the number of immunized persons.

Both these problems arise in plant pathology but their importance here is negligible. The annual epidemic of black rust of wheat is actually favoured by the rise of temperature in summer (cyclic disposition, p. 143). Hence it should decline again as the maximum summer temperatures are passed, but by that time the crops have been harvested. Conditions for the progressive immunization of stocks are even more unfavourable since, as we shall see in Chapter 4, acquired immunity is found only in very rare instances among plants.

(c) *A Decrease in the Vitality of the Pathogen as a Condition for the Decline of an Epidemic*

As the plant stock becomes ever more saturated by disease the susceptible individuals tend to be eliminated and the pathogen, therefore, must cope with more resistant plants in increasing numbers. But in the majority of cases passage through uncongenial hosts tends to weaken the pathogen (cf. Chapter 3); in particular, there is a lessening of (*aa*) its vitality, and (*bb*) its capacity for spore dissemination.

(*aa*) Reduction of vitality. If a strain of *Phytophthora infestans* be inoculated into the tubers of a resistant potato variety and then transferred successively to new tubers of the same variety it loses its vitality within a few weeks and dies out after five to eleven generations (K. O. Müller, 1936). Hence, pathogens of this type must become attenuated as the plant stocks are increasingly saturated by disease, leading to the decline of the progressive epidemic.

(*bb*) Regression in spore dissemination. As the difficulties of the pathogen increase, not only the quality or vitality of the spores, but also their number tends to diminish. With less luxuriant development it sporulates more and more sparsely and consequently the series, which has progressed in a positive direction during the rising phase of the epidemic, now becomes regressive: decreasing numbers of spores → decreasing doses of infection → decreasing severity of the disease → fresh reductions in spore numbers, &c., until a lower limiting value is reached. This quantitative reduction in spore dissemination is also enhanced by a topographical factor: because susceptible individuals are constantly eliminated they disappear, and thus the susceptible area becomes broken up and thinned out. Hence, the spore reservoir loses its density and its continuity and therewith its disseminative potency.

Therefore, when an epidemic among plants changes in character and subsides, both host and parasite contribute towards the event. The host alters in character, because, through the growing saturation of the stock by disease, only increasingly resistant individuals are left, and perhaps also because the general disease proneness of the stock diminishes. The pathogen also changes in character, since its infectivity and its chances of spore dissemination lessen progressively.

The interaction of these two factors, the selection of more resistant host individuals, and the consequent weakening of the pathogen, causes every epidemic to become less harmful as disease saturation proceeds. However, this recession of the parasite need not be genotypic, it may be purely phenotypic. If, therefore, the pathogen be carried to more favourable regions, the regressive development may again become progressive, activity and vitality may be restored, and the secular rhythm may begin all over again.

CHAPTER 3

THE PARASITIC ADAPTATION OF PATHOGENS

THE parasitic adaptation of a pathogen, like any other manifestation of life, is not a constant, an independent value, but is conditioned by internal and external factors; hence § 1: 'Innate, Constitutional Differences in the Parasitic Adaptation of Pathogens'; § 2: 'Changes in the Parasitic Adaptation of Pathogens' (p. 192); and § 3: 'The Conditions of Parasitic Adaptation of Pathogens' (p. 238).

§ 1. Innate, Constitutional Differences in the Parasitic Adaptation of Pathogens

In this section three questions will be discussed:

1. How great are the quantitative differences in the vigour and intensity of the capacity for parasitic attack among pathogens? ('Affinity, Aggressiveness, Pathogenicity');
2. What are the qualitative differences, i.e. the variations in scope of the capacity for parasitic attack? (2. 'The Choice of Host'; p. 179);
3. How are parasitic capacities distributed among the various systematic units of a pathogen? (3. 'Specialization of Pathogens'; p. 183).

1. *Affinity, Aggressiveness, Pathogenicity*

There is a tendency to regard the parasitic way of life as more advanced and the saprophytic mode as more primitive because the colonization of a host demands a number of special aptitudes in a pathogen which fit it for a parasitic career.

In the first place it must be chemotropically attracted to its host, i.e. it must be compatible with it and possess the corresponding tropism. Only then does the parasite react or make any attempt to invade the host. Thus, the conditions illustrated in Fig. 1 apply only to a compatible parasite-host relationship; for instance, if a rust spore strays on to a beech tree it dies without seriously attempting to infect it because the two partners are incompatible.

The number of pathogens attacking a given host species, for example, the number which attempt to colonize the host by inserting germ tubes through the stomata into the air chambers, may well be some dozen times more than the actual number of genuine parasites on the host (e.g. Cook, 1937). Thus, the zoospore germ tubes of *Plasmopara viticola* (downy mildew of vine) attack the most improbable hosts, on which they can never gain a foothold, e.g. Liliaceae, Polygonaceae, Labiatae, Leguminosae, Compositae, &c. (Arens, 1929). Further, within the genus *Vitis*, they attempt to colonize the foliage leaves of both susceptible and resistant species (Fig. 135). Therefore the mere fact of the penetration of a germ tube into a host does not necessarily imply successful infection.

Hence the 'affinity' between the phanerogams and the potentially parasitic micro-organisms is only slightly specific and its selective effect on the latter insignificant.

In the absence of affinity the pathogen does not attack the host but grows over it indifferently, as over a neutral substrate; the plant in question simply does not exist for the fungus.

Secondly, the pathogen must possess the capacity to invade, i.e. to infect its host, to inhabit or live in it, to overcome its resistance, to make use of it for its own nourishment, and to reproduce itself in or on it. This capacity is called aggressiveness and it is the essence of the parasitic mode of life.



FIG. 135. The penetration of *Plasmopara viticola* (downy mildew of vine) in the leaves of an uncongenial host species, *Vitis riparia* (1) and a congenial host species, *V. vinifera* (2). State of development after 3 days. Approx. $\times 900$. (After Arens, 1929.)

Aggressiveness is, therefore, manifested on the one hand by the facility with which an infection is established (aptitude for infection, efficiency of infection, or capacity to produce infection), and on the other hand by the facility with which a pathogen spreads in the host and makes itself at home. Criteria of aggressiveness are, therefore, as follows:

- (a) The numerical infection threshold (p. 38): a rust fungus which can establish itself by means of a single germinating spore, is more aggressive than a *Fusarium*, which can achieve the same end only by 10,000 spores (Fig. 38).
- (b) The time required for infection (p. 35): downy mildew of vine, which under optimal conditions enters into a stable parasitic relationship with the host in 2–3 hours, is more aggressive than a wound parasite of the *Valsa* group (p. 60), which takes several days even under the most favourable conditions.
- (c) The number of cases of disease: an isolate of *Pythium de Baryanum*, which under given external conditions causes disease in 100% of spruce seedlings, is ten times more aggressive than one causing only 10% infection under the same conditions.
- (d) The duration of the incubation period (i.e. the rapidity with which a parasite produces symptoms: *Puccinia dispersa* (brown rust of rye) is more aggressive at an air temperature of 20°C., at which the incubation period lasts only 6 days, than at 10°C., when it extends over about 15 days.

If aggressiveness be lacking the pathogen cannot succeed in its attempt

to colonize the host: the infection is abortive; *Plasmopara viticola*, for instance, possesses an affinity for both vine and lilac leaves but true aggressiveness only for the former, on which alone it succeeds in forming haustoria (Fig. 135, 2).

When an aggressive parasite spreads freely or its infection threshold is low, so that it 'takes' readily, it is called infectious or 'catching'; thus, the tobacco mosaic virus is more infectious than the *H*-strain of the potato X-virus group (p. 39).

In other cases, however, the meaning attached to the term 'infectious' is variable. A particular race (biotype 2) of dwarf or brown rust of barley, *Puccinia hordei* (syn. *Puccinia simplex*) establishes itself by single spore infections equally well on barley (*Hordeum vulgare*), bristle-pointed oat (*Avena strigosa*), and mouse or wall barley (*Hordeum murinum*); it is thus equally infectious on all three hosts. On barley (Hassebrauk, 1932), however, it produces the true brown rust with uredospores and teleutospores (primary host), on bristle-pointed oat only isolated pustules of uredospores after which it ceases growth (subsidiary host), and on wall barley merely necrotic spots which remain sterile (sub-infections). Hence in the terminology used in this book the aggressiveness of brown rust, i.e. its ability to colonize the host, is very different on the three species despite its uniform infectiousness.

The expressions 'aggressiveness' and 'infectiousness' describe the establishment of the parasitic relationship from the angle of the pathogen; from the angle of the host, conditions are different, because the host does not usually undergo colonization by the parasite without showing symptoms; it reacts and becomes diseased. This diseased state (Chapter 5) is certainly not 'intended' by the fungus, which 'desires' only to feed and reproduce itself at the host's expense. It is undoubtedly an 'unintentional' and often, moreover, an 'unwelcome' incidental effect that the host reacts to its presence and may even perish, leading under certain conditions to the termination of the life of the individual pathogen (Chapter 4, parasitic parasite-host pairs).

The ability of a pathogen to evoke disease is called its pathogenicity (from *pathos*, suffering). It is the measure of its parasitic efficiency or its parasitic effect; just as aggressiveness determines the infection of the host, so pathogenicity determines its disease reaction. If the ability to cause disease be lacking the parasite is apathogenic. Thus, the black moulds, e.g. *Cladosporium herbarum*, which often develop on cereals with foot rot, are aggressive since they are able to colonize and feed on the hosts, but they are apathogenic since their harmful effect is merely an insignificant drain of nutriment.

A by-product of pathogenicity, of the *pouvoir pathogène*, is the toxicity of the parasite, its poisonousness, its *pouvoir toxigène*. The pathological reactions of the host are evoked by poisons or toxins, which are often specific and can be isolated chemically and defined; they are secreted by the pathogen under particular conditions.

The parasitic efficiency of a pathogen is often termed its virulence (from *virus*, poison) instead of its pathogenicity, but unfortunately the former term is ambiguous.

In the wider sense, used primarily in medicine, the term virulence connotes a high aggressiveness and a high pathogenicity of the parasite, i.e. its total parasitic capacity or activity, in fact, its parasitic potential.

In a narrower sense, virulence is often used as the equivalent of pathogenicity; *Bacillus tetani*, for instance, is a bacterium highly virulent to man causing 100% mortality with an adequate dose of inoculum.

In another sense, virulence merely implies the ability of the pathogen to multiply in its host; in this sense tetanus bacilli are avirulent, being unable to spread in the interior of the infected organism although they are so highly pathogenic.

The interrelationships between aggressiveness, pathogenicity, and virulence are manifold.

Among the perthophytes (p. 60) pathogenicity is the pre-condition of aggressiveness, a highly pathogenic parasite of this group being at the same time highly aggressive.

On the other hand, in the biotrophic parasites aggressiveness is the pre-condition of pathogenicity; the host must first be colonized by the pathogen before it can become diseased. Quantitatively, however, their proportions are independent of each other: high aggressiveness may be combined with low pathogenicity and slight aggressiveness with high pathogenicity.

In medicine, measles is very infectious but only slightly pathogenic. In botany the nodule bacteria of the Leguminosae (Fig. 178) are strongly aggressive since they readily colonize the host, but virtually apathogenic since they cause no actual disease.

Anthrax infection in mice can be brought about by a single germ (p. 39), whereas 100,000 bacteria must be injected, *ceteris paribus*, into sheep. The end result, death, is the same in both cases, and therefore the causal organism is equally pathogenic to both hosts; on the other hand, it is termed more aggressive or 'more virulent' to mice on account of its lower infection threshold. *Fusarium vasinfectum* (cotton wilt) and *Fusarium cubense* (Panama disease of banana) are only slightly aggressive, having, like the other Fusaria (Figs. 38 and 39), a high infection threshold; conversely, they are highly pathogenic and usually lead to the death or at least to the sterilization of the plants attacked.

The ambiguity in the use of all these terms is itself a reflection of the endless diversity of the parasitic life, which can never be completely covered by any single term. The choice of the most felicitous turn of phrase to convey the facts of each particular case must therefore always be left to the discretion of the individual.

If a pathogen can feed only on living matter and hence cannot be cultured on an artificial medium in the laboratory, the botanist describes it as an obligate parasite or sometimes as an obligate-biotrophic parasite. Here belong, from the Archimycetes, *Synchytrium* (Fig. 187) and *Plasmodiophora*

(Fig. 128), from the Oomycetes the more highly evolved forms of the downy mildews such as *Plasmopara* (p. 103) and *Peronospora* (p. 37), from the Basidiomycetes the rust fungi or Uredineae, from the phanerogams the mistletoes, and finally all the viruses. Once these obligate parasites have killed their host they must either starve or complete their life-cycle by the production of resting spores.

The facultative or facultative-biotrophic parasites, in the botanical sense, can also maintain themselves saprophytically during the intervals when the host is absent, e.g. on plant material that has died or been killed by the parasite, and hence they can be grown in the laboratory in pure culture on artificial media. They comprise most of the plant pathogenic fungi and all the plant pathogenic bacteria.

Obligate parasites in the botanical sense are unknown in medicine, apart from the viruses, which can reproduce themselves only in tissue culture. In medicine the term obligate parasite is used for those micro-organisms which can be grown saprophytically on artificial media but which normally do not multiply outside the human body, e.g. tubercle bacilli and spirochaetes. Facultative parasites in the medical sense, such as *coli*-bacteria, can multiply both outside and inside the host.

Among the facultative parasites in the botanical sense the type of nutrition, whether saprophytic or parasitic, depends not only on the pathogen but also on the host. Intestinal bacteria of the *coli* group are harmless commensals so long as they live only in and on the intestinal contents, but if a susceptible condition of the host permits their attack on the mucous membrane, they become parasites and produce typhoid diseases. Nevertheless, even the layman recognizes an essential difference between the parasitic and the saprophytic modes of existence of a pathogen, as shown by the subtle distinction in German between *Ersticken* ('dote') and *Vermorschen* (rot) in the biological decomposition of wood.

The expression *Ersticken* of wood has indeed a dual meaning in German. On the one hand, it is applied to that form of decay of sawn timber in which the hyphae of the fungus, e.g. *Coniophora cerebella*, are not immediately visible to the naked eye, so that the wood seems to have deteriorated of its own accord from lack of air.

On the other hand, however, *Ersticken* is applied primarily to fungal decay of living wood, especially in felled but still living trunks. Felled trunks do not die forthwith, but after passing the winter covered with bark growth is renewed in the following spring, fresh shoots are put out, and death ensues only with the onset of desiccation. However, when felled their vitality is reduced, and consequently they are attacked by specific parasites, e.g. beeches by *Polystictus* spp., spruces and firs by *Lenzites heteromorpha*, the cause of 'flamey' wood, resulting in their becoming 'dotey' (Gäumann, 1930 and 1936).

The layman uses the other term, *Vermorschen*, for the biological decomposition of dead wood, which is affected by specific fungi; thus, *Lenzites heteromorpha* is a fungus that causes 'dote' in still living wood whereas

L. abietina is a fungus that causes rot in dead wood, e.g. in spruce and fir timber. This distinction applies, however, only to the primary stages of infection; with the death of the above-mentioned felled trunks the mode of existence of *L. heteromorpha* also changes during the summer from 'Ersticken' to 'Vermorschen'.

2. The Choice of Host

Parasitic micro-organisms and viruses are not able to colonize any and every kind of plant, but as a rule only a limited number of species, and in such cases a given parasite is dependent for its development on particular host species. This phenomenon is known as their 'choice of host' and the range of their host species or food plants is termed their host circle or host spectrum.

The expression 'choice of host' is not a happy one. A choice presupposes free decision within the limits of an offer, whereas the selection of a host is in most cases negatively circumscribed, an involuntary 'choice' of what the parasite, with its restricted faculties, can attack; it has the choice only of entering by the single door that it can open or perishing. Thus, it is the pathogen that is chosen or tolerated, not the host. The expression 'choice of host', therefore, makes a virtue of necessity, but it would be inexpedient to replace it by another to-day.

The range of host choice, the width of the spectrum, varies greatly in extent with different pathogens. It is very wide in a number of opportunist and debility parasites. Thus, *Botrytis cinerea* (Fig. 1) can infect the most varied parts and organs of thousands of species of the most diverse and unrelated genera, families, and orders in greenhouse and field, provided only that they are in a susceptible, i.e. an enfeebled condition. In propagation frames that are inadequately ventilated this fungus attacks seedlings of practically all cultivated garden plants. It also colonizes stems, e.g. begonias, cucumbers, potatoes, &c., or fruits, e.g. pears, apples, tomatoes, oranges, dahlia fruits, grapes (grey mould), &c., and in addition edible and flower bulbs, potato tubers, and so forth. Parasites of this kind, whose host choice is almost unlimited, are called omnivorous or all-devouring.

Conversely, with pathogens that attack plants in full vigour the host circle is never so indeterminate but always exhibits certain limitations. If the parasite colonizes hosts of various genera or families it is termed plurivorous, multivorous, pleophagous, polyphagous, or euryxenic. These terms are not all exact synonyms, but here again the shade of meaning is best left to the individual's feeling for words.

For example, the 'whorl' fungus *Verticillium albo-atrum* is plurivorous since it causes a wilt disease of over 70 plant genera, forest and fruit-trees, shrubs, root crops (Figs. 244 and 285), vegetables, weeds, and so on, in which practically all the isolates tested have been found to be transmissible to every other host (Donandt, 1932).

Corticium vagum (Fig. 58), a lower Basidiomycete which, under several names, frequently *Rhizoctonia solani*, in all likelihood attacks well over

200 species of the most heterogeneous families of monocotyledons and dicotyledons (Braun, 1930), is thus also plurivorous and gives rise to all kinds of pathological effects, e.g. potato stem canker and black scurf of the tubers, black leg of beets, and damping-off of conifer seedlings.

On the other hand, the host circle of *Puccinia graminis tritici* (black rust of wheat) is narrowly circumscribed. Of the thousands of species available it can attack only those of the single genus *Triticum* (wheat); it is confined to the genus *Triticum* and specialized in relation to it. Pathogens of this type are called stenoxic implying 'a narrow host range'.

Narrow and wide, however, are relative conceptions, dependent on the point of view. Anyone accustomed to *Botrytis cinerea* will find *Corticium vagum* specialized, whereas to anyone accustomed to working with races of black rust it will seem amazingly plurivorous.

When two host spectra overlap, the hosts in common are known as collective hosts; thus, in Fig. 136, the sugar maple is a collective host for the mistletoe of broad-leaved trees and for that of firs. On collective hosts there is always the chance of mixed infections.

Hosts belonging only to one circle may serve as differential hosts. Passage through differential hosts exercises a selective or filtering action by resolving the components of a mixed infection. Thus, a mixed growth of mistletoe on the sugar maple can be resolved into its two components even by one passage through the apple tree which is a differential host for the mistletoe of hardwoods or through a silver fir which is a differential host for the fir mistletoe. Differential hosts that show especially characteristic symptoms are used as test plants on account of their ready response.

The limits of host choice are, like the range, more or less characteristic for different pathogens.

In the case of *Phytophthora infestans* the boundaries of the host circle are obscure and indeterminate. Under extreme conditions in the laboratory it can infect seedlings, e.g. of beech, maple, and conifers, so that at times it is not easy to decide whether or not a certain host from the periphery of the spectrum could be attacked in a 'natural' environment.

On the other hand, the delimitation of the host circle is very sharp in the stem blister rust of Scots pine (Fig. 52, *Cronartium asclepiadeum*). In the uredospore and teleutospore phase it attacks, apparently at random, the most widely separated flowering plants, such as certain members of the Ranunculaceae (*Paeonia* spp., e.g. *P. officinalis*), Scrophulariaceae (e.g. the marsh lousewort, *Pedicularis palustris*), Balsaminaceae (e.g. balsam, *Impatiens balsamina*), Tropaeolaceae (e.g. a nasturtium, *Tropaeolum majus*), Asclepiadaceae (e.g. swallow-wort, *Vincetoxicum officinale*), &c. But it attacks only these species and not their nearest relatives. In contrast to *Phytophthora infestans*, therefore, the boundaries of *Cronartium asclepiadeum* cannot be experimentally widened by subjecting it to unusual conditions.

There are, of course, many transitional stages between these two extremes of vagueness and sharp definition. *Phymatotrichum omnivorum*, the conidial

phase of the dreaded Texas root rot in North America, is parasitic on the underground organs of no less than 1,708 dicotyledons and gymnosperms but, strangely enough, on not a single monocotyledon, for instance, on not one of the grasses (Ezekiel and Fudge, 1938). Thus, on the one hand, there is the wildest pleophagy and, on the other hand, an unexpectedly sharp delimitation.

The host spectra may be homogeneous or heterogeneous in structure, both qualitatively, in relation to the systematic affinity of the hosts, and quantitatively, in relation to the aggressiveness of the pathogens.

In host spectra which are homogeneous because of the systematic affinity of the hosts, the several host species are usually closely related. Thus, *Phytophthora infestans* (late blight of potato) attacks the most divergent Solanaceae but occurs spontaneously only on this family, i.e. a systematically uniform group, so that any new hosts can be predicted with fair assurance.

Similarly ergot species, e.g. *Claviceps purpurea* (Fig. 81) and the choke fungi (*Epichloë* spp.) attack only grasses; *Epichloë typhina* attacks the European grasses and *E. bambusae* the Bambusaceae, but no *Carices* or other phanerogams. Rust fungi of the genera *Phragmidium* and *Gymnosporangium* (e.g. cluster cup rust, p. 86) colonize only Rosaceae, so that if one finds a leaf bearing a parasite of either of these two genera it must belong to a rosaceous plant.

In these cases, therefore, the systematic relationship of the hosts is also accompanied by a physiological or biological relationship, which shows itself in a similar reaction to infection by certain pathogens (Moritz, 1932; Buhr, 1937). The origin of these positive linkages has been sought, *inter alia*, in the protein kinship of the hosts. To some extent this may be correct but it must not be overlooked that the protein relationship is only one component amongst many and that, serologically, only those proteins are involved which react to the particular technique employed; this is also true for the carbohydrate relationships.

On the other hand, the host spectra of *Corticium vagum* and *Verticillium albo-atrum* are heterogeneous and unconnected. Both fungi, like *Cronartium asclepiadeum*, pass to all possible hosts (p. 180).

No study has yet been made of the reasons for this seemingly arbitrary aggressiveness. For instance, it is completely unknown why our native *Cronartium asclepiadeum* should be able to colonize and feed on foreign angiosperms such as *Nemesia versicolor*, a South African species of the Scrophulariaceae, *Verbena erinoides*, a Peruvian species of the Verbenaceae, and *Grammatocarpus volubilis* a Chilean species of the Loasaceae. All these are, without exception, experimental new hosts which the fungus cannot have encountered previously. It is also unknown why it attacks only these and no others, not even the most closely related genera and species.

Usually, a relationship exists between the constitution and the range of the host spectrum inasmuch as wide host spectra are often heterogeneous

whereas narrow host spectra are mostly homogeneous. There are, however, many exceptions, e.g. *Cronartium asclepiadeum*, whose host spectrum is narrow but heterogeneous.

In host spectra that are homogeneous as regards the virulence of the pathogen, the latter possesses the same aggressiveness and pathogenicity towards all its hosts. This very seldom occurs in the plant world but *Verticillium albo-atrum* (p. 179) may, perhaps, be such an exceptional case.

As a rule the virulence of the pathogen within its host spectrum, more especially towards the periphery, is graduated, the host spectrum thus being quantitatively heterogeneous. It is true that under certain conditions the brown rot fungus of cherries, plums, &c., can also attack apricots (Table XXI), but only on the former does the resulting disease pursue the specific and devastating course of die back of the branches. The tobacco ring spot virus is lethal to beans *Phaseolus vulgaris*, whereas on tobacco it merely induces local necroses (Fig. 223), and on sweet clover (*Melilotus officinalis*) sometimes grown in rotation with tobacco, no symptoms whatever; sweet clover is, therefore, a symptomless carrier.

TABLE XXI

The morphological and biological differentiation of two Sclerotinia spp. on stone fruits. (After Aderhold and Ruhland, 1905)

Species	Dimensions of asci	Dimensions of ascospores	Principal hosts	Subsidiary hosts
<i>Sclerotinia cinerea</i> .	(μ) 90–110 \times 6–8	(μ) 6–9 \times 3–5	Cherries, plums, peaches, &c.	Apricots
<i>Sclerotinia laxa</i> .	120–150 \times 8–12	11–14 \times 5–7	Apricots	Cherries, plums, peaches, &c.

This phenomenon of a plurivorous parasite being able to attack certain species of its host circle more mildly and others more severely is known as its specific pathogenicity for the particular host species. The most-favoured species of host is called the principal host, the less compatible ones the subsidiary hosts, while subsidiary hosts that are attacked only exceptionally or under conditions seldom realized are termed occasional hosts.

However, as in so many biological matters, the terminological usage has been overwhelmed by the facts, so that the gradation between principal and subsidiary hosts must also be considered from other aspects. For example:

- (a) From the economic point of view. The potato (*Solanum tuberosum*) is deemed to be the principal host of potato wart disease (*Synchytrium endobioticum*) if only for the reason that economically it is the important one. This is irrespective of whether it is more severely or more mildly attacked than the remaining species of the host circle,

Solanum Commersonii, *S. Dulcamara*, *S. guaranticum* (syn. *S. Cha-coense*), *S. Jamesii*, *S. Lycopersicum*, *S. nigrum*, &c., and regardless of the fact that historically it is not even the original host of potato wart (p. 154).

- (b) From the point of view of frequency of incidence. The swallow-wort (*Vincetoxicum officinale*) is known in Central Europe as the chief alternate host of stem blister rust of Scots pines, because it is by far the most common host. The others, irrespective of whether they are more or less susceptible, are only 'subsidiary hosts' for the transmission of the disease.
- (c) From the point of view of the parasite's fecundity. On the black currant (*Ribes nigrum*) the blister rust of Weymouth pine (*Cronartium ribicola*) produces some 2,600,000 basidiospores per sq. cm. of infected leaf surface, and on the ornamental golden currant (*Ribes aureum*) about 900,000, but under the same external conditions on two North American wild species, *R. setosum* and *R. lacustre*, only 120,000 and 45,000 or so, respectively (Taylor, 1922). *R. nigrum* and *R. aureum*, therefore, rank among the principal hosts and *R. setosum* and *R. lacustre* among the subsidiary (alternate) hosts of the blister rust.
- (d) From the point of view of the life-cycle of the parasite. Host species serving the parasite for the production of its perfect state (i.e. in fungi, that form of fructification in which nuclear fusion or karyogamy occurs), are known as primary hosts, whereas those used merely for the development of the imperfect states in fungi (usually the asexual spore forms, conidia, uredospores, &c.) are known as secondary hosts. Barley is the primary host of the dwarf or brown rust (*Puccinia hordei*) mentioned on page 176, because this fungus develops teleutospores on it and therewith completes its life-cycle, whereas the bristle-pointed oat is merely a secondary host, on which the fungus only produces uredosori. The various mid- and south-European *Quercus* spp. are primary hosts of the oak mildew (*Micro-sphaera alphitoides*), because the fungus forms its perithecia on them, whereas *Fagus sylvatica* and *Castanea sativa* are merely secondary hosts because on them the fungus produces only conidia.

3. Specialization of Pathogens

Originally it was assumed that each morphologically distinct species of pathogen represented both a morphological and a biological entity, and more especially that all the individuals of the species would possess approximately the same parasitic abilities. To-day there are only a few micro-organisms that conform to this expectation, e.g. downy mildew of vine (*Plasmopara viticola*) and the agents of club root (Fig. 128), potato wart (Fig. 187), and potato powdery scab (*Spongospora subterranea*).

In the overwhelming majority of cases the pathogens have shared the fate of their hosts but in even greater measure. With the refinement of morphological and biological analysis the Linnaean host species were split

into Jordanian species and ultimately into sibs and local races. In the same way the refinement of biological experimental technique from about 1890 onwards led rapidly to a dissociation of the Linnaean species into smaller bio-systematic units, form-groups or form-‘bundles’, distinguishable from one another by their host selectivity and often, also, by minor peculiarities of structure. They were termed biological species or *formae speciales* (singular *forma specialis*, abbreviated to f. sp., mostly followed by the genitive of the primary host).

Thus, the mistletoe (*Viscum album*) comprises at least three biological species, the hardwood mistletoe (f. sp. *mali*), the fir mistletoe (f. sp. *abietis*), and the pine mistletoe (f. sp. *pini*).

Of these the deciduous tree mistletoe (*V. album* f. sp. *mali*) has the widest host range, inhabiting every possible hardwood species, but only these; Tubeuf (1923) enumerates no less than thirty-six host genera. It can also parasitize individuals of its own kind, e.g. male plants on female plants, giving rise to the mistaken impression of mistletoe bushes with both male and female flowers. Possibly it may even subdivide into biologic races, because the mistletoe of the lime tree seems to have a specific capacity for infecting limes and the poplar mistletoe a similar capacity for attacking poplars.

The fir mistletoe (*V. album* f. sp. *abietis*) has *Abies* spp. for primary hosts; in Switzerland the silver fir (*A. pectinata*), in Greece *A. cephalonica*, in the Caucasus *A. Nordmanniana*, and so forth. It does not pass over to pines but does attack the North American silver maple (*Acer saccharinum*) (syn. *Acer dasycarpum*), and also *A. rubrum* and the Japanese larch (*Larix leptolepis*). Hence, the two *Acer* spp. are collective hosts for the mistletoes of hardwood trees and firs.

The pine mistletoe (*Viscum album* f. sp. *pini*), occupies *Pinus* spp. as its primary host; it occasionally attacks spruce (*Picea excelsa*), *Larix leptolepis*, and sallow or goat willow (*Salix Caprea*), but never attacks other deciduous trees or silver fir. *Larix leptolepis*, therefore, serves as a collective host for fir mistletoe and pine mistletoe, and *Salix Caprea* as a collective host for the mistletoes of broad-leaved trees and pines, but no collective host is yet known for all three biological species. Fig. 136 depicts graphically the three overlapping host circles.

As an example of biological species from the world of micro-organisms the powdery mildew of clover may be instanced (Table XXII). This comprises at least seven biological species; the one on red meadow clover, f. sp. *trifolii pratensis* Salmon is relatively polyphagous and even colonizes members of different sections, whereas f. sp. *trifolii repentis* Hammarlund is limited to white clover and is, therefore, strictly specialized.

However, Table XXII shows also the practical difficulties of this classification, since the red meadow clover not only harbours (as principal host) the above-mentioned f. sp. *pratensis*, but also (as subsidiary host) the fae. sp. *trifolii procumbentis* and *trifolii medii*. If desired, the components of a mixed infection may be separated by passages through differential hosts.

TABLE XXII

The specialisation of Erysiphe Martii within the genus Trifolium. (After Blumer, 1933)

Inoculum from		Tr. procumbens	Tr. agrarium	Tr. repens	Tr. hybridum	Tr. medium	Tr. pratense	Tr. incarnatum
applied to		<i>f. sp. trifolii</i> procumbentis <i>Blumer</i>	<i>f. sp. trifolii</i> agrarii <i>Hammarl.</i>	<i>f. sp. trifolii</i> repentis <i>Hammarl.</i>	<i>f. sp. trifolii</i> hybridi <i>Hammarl.</i>	<i>f. sp. trifolii</i> medii <i>Blumer</i>	<i>f. sp. trifolii</i> pratensis <i>Salmon</i>	<i>f. sp. trifolii</i> incarnati <i>Neger</i>
Section	Species							
Chronosemium	Tr. procumbens	+				—	—	
	Tr. agrarium	+	+	—	—	—	—	
	Tr. badium	—				—	—	
Euamoria	Tr. montanum	+				—	+	
	Tr. repens	—	—	+	—	—	—	—
	Tr. hybridum	+	—	—	+	+	+	
Lupinaster	Tr. alpinum						—	
Galearia	Tr. fragiferum	+				—	+	
Eulagopus	Tr. medium	—	—	—	?	+	+	
	Tr. pratense	+	—	—	—	+	+	—
	Tr. alpestre	+				+	—	
	Tr. rubens					—	—	+
	Tr. incarnatum						—	
	Tr. arvense	—	—	—	—	—	—	

From about 1915 onwards the micromanipulative technique of single spore or single cell culture enabled workers to analyse biological species into their ultimate systematic units, single cell cultures or isolates.

These are assumed to be pure when they are clones, i.e. vegetative progeny of an individual, e.g. a cell. In the biological sense these clones are individuals which, owing to the nature of microbial reproduction, can be grown and replicated in an indefinite number of transplants, 'cultures',

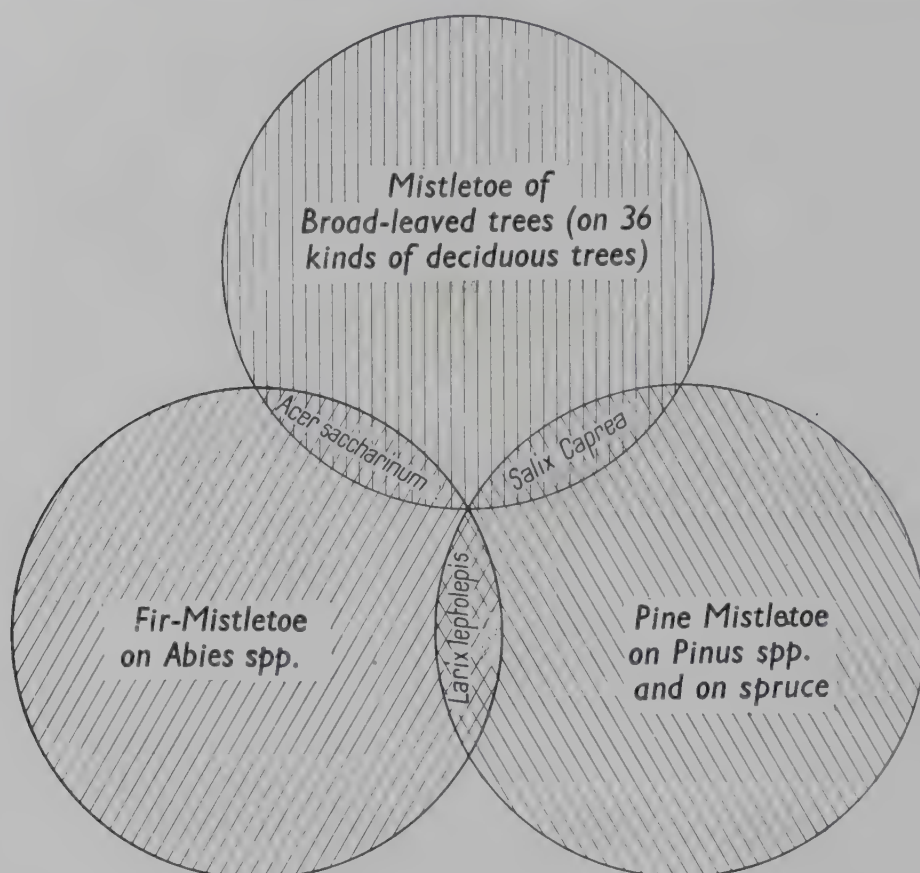


FIG. 136. The overlapping of the host ranges of the three biologic species of mistletoe (*Viscum album*).

or 'specimens'. The offspring of the sexual propagation of these clones are, of course, no longer biologically identical with their parents and thus represent new clones.

However, as we shall see later in the section on nuclear segregation, not every 'pure culture' is really pure merely by reason of its origin in a single spore or a single cell. Single spore and single cell cultures are, if anything, more likely to be genetically impure if the original spore be multicellular or if the original cell be multinucleate. In such cases the cultures must be further analysed until the genetically pure individuals are obtained.

These vegetatively propagated individuals (pathogenic clones) constitute the modern systematic units of the parasitologist. They are called races, strains, forms, or biotypes, and designated by serial numbers, letters, the name of a typical host, or their geographical source, &c.

Unfortunately, race here means something different from the races of phanerogamic or host taxonomy. In the latter the race is a population of

individuals, e.g. a 'local race', whereas, among the pathogens, it is a vegetatively propagated individual, i.e. a clone.

The races of the various pathogens differ from one another in numerous ways, the criteria sometimes being morphological and sometimes biological.

Predominantly morphological races of about equal parasitic efficiency occur in the fungus causing black spot of rose. True, in the periphery of its host spectrum a careful examination will reveal slight differences, e.g. the capacity of a single clone to infect *Rosa arvensis* and *R. glauca* (Table XXIII). However, their host choice is essentially identical; severe attack on cultivated roses, milder attack on the Caninae, e.g. on *R. pomifera* and *R. eglanteria*. Their physiological requirements, e.g. temperature, are also alike. The only dependable criteria are the conidial dimensions: the races differ morphologically just as people differ in stature.

Races (i.e. actual individuals) differing only in cultural habit have been observed, for example, in numerous smut clones. On the same medium these show slight deviations in growth, consistency, colour, and margination of the colonies, as well as in temperature requirements (e.g. Rodenhiser, 1928). All of these minutiae permit immediate recognition of individual isolates in the routine of daily laboratory work but are difficult to explain verbally to other people.

Nutritional-physiological races which are morphologically and parasitically identical occur in ergot of rye (*Claviceps purpurea*). Certain strains thrive particularly well on malt agar on which they can be distinguished by their copious production of mycelium and conidia, whereas others grow sparsely if at all on this medium and prefer cane sugar-asparagin-agar (Krebs, 1936). They also differ to some extent in their metabolism, e.g. in their capacity to produce ergotamin.

Differences in aggressiveness only have been observed. For instance, *Pythium de Baryanum* includes both highly aggressive strains which cause 100% root rot disease of spruce seedlings, and purely saprophytic strains which induce no infection under the same external conditions and are thus non-aggressive (in medical terminology avirulent). In such groups of parasites, therefore, it is not enough to establish the presence of a given pathogen; in addition, the actual aggressiveness and pathogenicity of its isolates must be determined.

In general, however, the various biotypes are distinguishable not by one but by a combination of these characters. Rudloff (1935), for instance, isolated from leaves of German and Swiss apple varieties 172 single spore cultures (i.e. actual individuals) of the apple scab fungus (*Endostigme inaequalis* = *Venturia inaequalis* = *Fusicladium dendriticum*). No two of these were alike in habit and rate of growth, colour and structure of the mycelium, production of conidia, conidial dimensions, aggressiveness, and host selectivity. Of 473 single spore cultures from different parts of Germany examined by M. Schmidt (1937) at least 448 were recognizably distinct, i.e. scarcely two among them were identical; a single leaf of the variety 'Beauty of Boskoop' carried no fewer than eight biotypes (Schmidt, 1935).

TABLE XXIII

The morphological and biological segregation of Diplocarpon rosae (black spot of rose). (After Frick, 1943)

Fungus strain No.	Colour of cultures	Summer conidia		Severity of attack* on						
		Length μ	Width μ	Étoile de Hollande	W. E. Chaplin	Rosa lutea	Rosa arvensis	Rosa glauca	Rosa pomifera	Rosa eglanteria
6	dark brown with ochra- ceous zones	18.2 \pm 1.8	6.7 \pm 0.6	14.2	13.5	2.0	0	0	3.6	8.9
20	dark brown or ochra- ceous	20.3 \pm 2.2	5.6 \pm 0.9	9.4	10.6	2.2	0	2.9	4.8	7.7
25	ochraceous	16.9 \pm 1.5	5.4 \pm 0.6	11.3	13.2	2.5	1.9	5.1	3.6	9.4

* Average number of lesions per pinnate leaf.

Table XXIV illustrates some cultural differences in three of them and Table XXV gives the reaction of such biotypes to test varieties, i.e. their host selectivity.

TABLE XXIV

Some cultural characters (on cane sugar-peptone-agar) of three biotypes of Endostigme inaequalis isolated from a single leaf of var. 'Beauty of Boskoop'. (After M. Schmidt, 1935)

<i>Biotype no.</i>	<i>Margin of the colony</i>	<i>Colour of the aerial mycelium</i>	<i>Structure of the hyphae</i>	<i>Conidial formation</i>	<i>Oidial production</i>
1	slightly stellate	dark grey	predominantly smooth	none	very profuse
4	slightly stellate	pale grey	slightly undulating	fairly profuse	poor
5	smooth	dark grey	smooth, lateral excrescences	none	very poor

TABLE XXV

Host choice of seven biotypes of Endostigme inaequalis. (After M. Schmidt, 1937)

<i>No.</i>	<i>Inoculated on</i> <i>Biotype of</i>	<i>Winter Golden Pearmain</i>	<i>Cox's Orange</i>	<i>Lands-berger Reinette</i>	<i>Berner Rosen- apfel</i>	<i>Beauty of Boskoop</i>	<i>Graven- steiner</i>	<i>Jakob Lebel</i>
M 1	Winter Golden Pearmain	+	+	+	+	—	+	+
M 28	Cox's Orange	+	+	+	+	+		+
M 31	Landsberger Reinette	+	+	+	+	—	+	
M 40	Berner Rosenapfel	+	+	+	+		—	+
M 46	Beauty of Boskoop	+	+	+	—	+	—	+
M 51	Gravensteiner	+	+	+	+		+	+
M 59	Jakob Lebel	(+)	+	+	+	+	+	+

+ Normal infection; (+) sub-infections; — no infection.

Similar conditions obtain in *Helminthosporium gramineum* (barley leaf stripe disease). On the basis of some 1,200 single spore cultures from the Old and New Worlds, Christensen and Graham (1934) provisionally distinguished over 125 separate races, differing from one another in their growth and colour on certain media (black, purple, grey, light or dark red, olive-green, &c., Fig. 137), in the septation and dimensions of their conidia (averaging in one race $101.4 \pm 0.21 \mu$ in length, and in another only $65.7 \pm 0.74 \mu$), and in their aggressiveness (incidence of disease for one race 0 and for another 76%). Moreover, the number of these races can be multiplied almost indefinitely by further isolations. *Botrytis cinerea* also shows extensive segregation into strains differing from one another, e.g. in their tendency

to the formation of mycelium, conidia, or sclerotia, in their growth habits on differential media, in their conidial dimensions, and in their aggressiveness, pathogenicity, and host choice (Paul, 1929). Such strains are called morphological-physiological races.

In the form-genus *Fusarium* (causal agents of wilt diseases, dry rots, &c.) the number of members and the extent of morphological and biological segregation are so great that classification of the individual isolates in discrete systematic units on the lines of orthodox taxonomy is precluded and they can be dealt with only by experienced specialists who group them around certain averages as 'forms of the type *Fusarium* . . .' (Wollenweber, 1926-35, 1931).

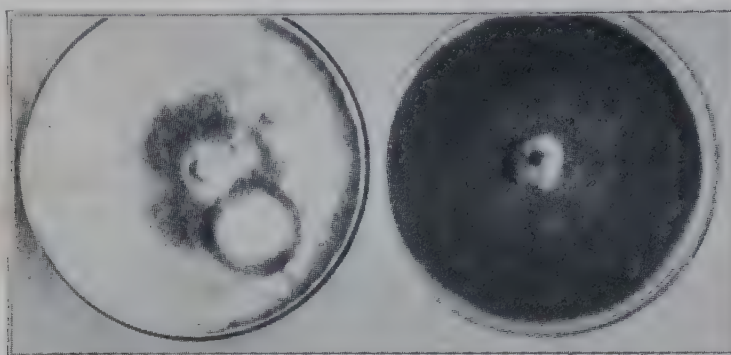


FIG. 137. Two races of *Helminthosporium gramineum* isolated from the same barley leaf. Left, a white race with a saltation sector at lower right; right, a red race. Approx. $\times \frac{1}{3}$. (After Christensen and Graham, 1934.)

In view of its practical importance in breeding for immunity the most extensive analysis of biotypes has been carried out on the rusts, smuts, and powdery mildews of annual agricultural plants, because here a new biotype of a pathogen may, in certain circumstances, be the equivalent of a new pathogen (p. 153). As in apple scab the host 'varieties' are merely portions of a tree that are vegetatively propagated as slips and grafted on to various rootstocks, so among annual cultivated plants the host sibs are no longer defined in the botanical sense but only sub-botanically as 'selections'.

Table XXVI indicates how a population analysis is carried out by means of standard varieties. Stakman *et al.* (1935) provide keys for the determination of no fewer than 144 biotypes of black rust of wheat (*Puccinia graminis tritici*).

Such biotype keys are most useful at the time of their compilation; if in 50 years' time the test varieties are no longer pure or if they never were pure in the genetic sense, the keys will possess only historical value. On the other hand, the existence of such extreme biological specialization among parasites proves that agricultural selections differing from one another so minutely that the distinctions can scarcely be expressed in morphological terms still constitute biological entities.

This is important in relation to legal protection in plant breeding. The law of many countries is defective inasmuch as inventions of a relatively

low order of originality in the technical field enjoy State, i.e. Patent Office, protection, whereas new selections of agricultural and horticultural plants, the outcome perhaps of decades of work and capital investment, are almost unprotected against piracy.

TABLE XXVI

Key for the determination of seven biological races of loose smut of wheat (*Ustilago tritici*). (After Grevel, 1930)

Wheat varieties serving as differential hosts	Biotype
Grüne Dame susceptible	
Rimpaus Roter Schlanstedter susceptible	
Grüne Dame × Rimpaus Roter Schlanstedter, strain 9,500, susceptible .	3
Grüne Dame × Rimpaus Roter Schlanstedter, strain 9,500, resistant .	7
Rimpaus Roter Schlanstedter resistant	2
Grüne Dame resistant	
Rimpaus Roter Schlanstedter susceptible	
Grüne Dame × Rimpaus Roter Schlanstedter, strain 9,500, susceptible	5
Grüne Dame × Rimpaus Roter Schlanstedter, strain 9,500, resistant .	
Grüne Dame × Rümkers Sommerdickkopf, strain 9,357, susceptible .	4
Grüne Dame × Rümkers Sommerdickkopf, strain 9,357, resistant .	1
Rimpaus Roter Schlanstedter resistant	6

To remedy this condition the Reich Food Board in 1934 instituted a Register of Varieties for the German State in which only tested authentic varieties were to be included; these were to be the only source of commercial seed. In certain doubtful cases differential testing with highly specialized parasitic races, e.g. rust biotypes, allows old varieties falsely described as new and given unfamiliar names to be recognized even if suspicions based on other characters might scarcely be upheld in law. The delicacy of this test is comparable to the serological proof of identity.

Finally, the example of the black rust of cereals and grasses may serve to elucidate the modern classification of a pathogen. The black rust was originally described by Persoon (1797) on 'cereals' as *Puccinia graminis*. During the nineteenth century it was found on more and more species of cereals and grasses, so that its known host range comprised over 100 different Gramineae. It also aroused great economic interest because in mild rust years it caused crop losses of some 20%, and in severe rust years of 50% and upwards; it is estimated that for Germany alone the annual loss averages about 200 million RM.

In the year 1896 Eriksson and Henning divided it into six biological species, f. sp. *tritici*, black rust of wheat with *Triticum* as primary host, f. sp. *secalis*, black rust of rye with *Secale cereale* as primary host, f. sp. *avenae*, black rust of oats with *Avena* as primary host, &c. These biological species also show minor morphological differences; thus, the uredospores of the above forms are all of approximately the same width (13–18 μ), but the uredospores of f. sp. *secalis* are definitely longer than those of the two other forms, namely, about 31–44 μ as against 21–35 μ and 23–35 μ respectively.

Since the beginning of this century our knowledge of black rust has

increased rapidly in scope and content; thus Lehmann, Kummer, and Dannemann's volume *Der Schwarzrost* (1937) contained 584 pages and cited roughly 1,900 references. Eriksson's biological species, *formae speciales*, or special forms were gradually subdivided into biological micro-species, races, and biotypes. For instance, f. sp. *tritici* was split into 300 biotypes and the end of this progressive analysis is not yet in sight and may never be reached. Some of these biotypes exhibit in turn small morphological differences which sometimes are almost as large as those between the special forms. Thus the uredospores of black rust of wheat, biotype 1, on a congenial host are $34.8 \pm 0.22 \mu$ long and $20.2 \pm 0.08 \mu$ wide, those of biotype 29, again on a suitable host, are only $28.7 \pm 0.19 \mu$ in length and $19.4 \pm 0.07 \mu$ in width, i.e. shorter and more rounded.

In consequence of this splitting the nomenclatural categories hitherto adopted would become too detailed for ordinary use, and for the sake of uniformity must be discarded. The old *Puccinia graminis* constitutes a form cycle, a group species, a collective or Linnaean species. The earlier special forms, *tritici*, *secalis*, &c., are raised to micro or Jordanian species, and in order to obviate new names are given tertiary names (a practice generally decried), namely, *P. graminis tritici* Eriksson, *P. graminis secalis* Eriksson, &c.; by this it is understood that *P. graminis tritici* Eriksson is a microspecies belonging to the form cycle of *P. graminis*. The systemic position of the individual biotypes, which economically are the centre of interest, is not affected by these changes; they constitute the permanent biological units of parasitology.

§ 2. Changes in the Parasitic Adaptation of Pathogens

The parasitic adaptation of a pathogen is not invariably constant. Changes may depend upon (a) internal and (b) external causes.

(a) Internal causes. It was long believed that single spore and single cell cultures of micro-organisms remained constant for any number of generations or sub-cultures, and this constancy served as proof of the purity of the cultures (theory of constancy of pathogen corresponding to constancy of host). The technique of micromanipulation first indicated that, owing to inner causes, micro-organisms could undergo sudden irreversible changes in their parasitic adaptation.

These internal causes may be of a vegetative nature, and (subsection 1) a matter either of nuclear dissociation (segregation), or (subsection 2) of nuclear association. In both cases the abrupt change is known as saltation, and its products as saltating clones or saltants.

Again, the inner causes may be of a sexual nature and (subsection 3) a matter of sexual nuclear association, following the pairing of opposite sexes (nuclear phase change).

Finally, the internal causes may be of a genetic nature and lead to a true genotypic alteration in the pathogen, namely, (subsection 4) as a result of hybridization, or (subsection 5) of true mutation (mutants).

(b) External causes. The genetic constitution of a pathogen does not

limit it to a fixed behaviour pattern, but rather circumscribes the zone of flexibility or sphere of modification within which the organism can react to its external environment. Within this zone of flexibility the immediate condition of the parasite and the host are decisive. It is the vitality and the parasitic disposition of the pathogen, and the disposition of the host to serve as a host, which determine the occurrence and the development of the parasitic relationship. Hence the changes occurring within this sphere of modification are phenotypic in nature, and consequently reversible. As a rule they are not sudden or discontinuous but gradual and continuous.

These external causes may be pre-infectional and modify the vitality of the parasite even prior to infection. The chief influences are: (subsection 6) pre-infectional nutrition of the pathogen, and (subsection 7) the modification of its vitality by the previous host (passage problems).

On the other hand, external causes may first operate during and after infection. In these cases the main influences are the co-existing microflora, the co-existing influence of mixed infections (subsection 8), and the surrounding temperature (subsection 9).

The influence of the amount of inoculum or of the severity of an infection on its course (p. 42) will not be discussed since the parasitic adaptation of the pathogen is unaffected by these although, as a rule, experience confirms the adage 'many hounds mean death to the hare'.

1. *Changes in Parasitic Adaptation due to Nuclear Dissociation (Segregation)*

In a large number of phytopathogenic fungi the cells of the hyphae are multinucleate. This phenomenon may be due to different causes: either (a) the spores that gave rise to them were multinucleate, or (b) the spores (zoospores, conidia, ascospores, &c.) were uninucleate but nuclear division without wall formation occurred during germination, or (c) the multinucleate condition arose by association of nuclei from different hyphae (to be discussed later). In certain fungi, e.g. *Phytophthora infestans* with its aseptate, multinucleate, *coenocytic* hyphae, all three possibilities are present together: if the zoosporangia (conidia) germinate directly, producing germ tubes, case (a) occurs, germination of the zoospores forms case (b), and hyphal anastomosis case (c).

If all the nuclei are genetically alike then the multinucleate condition of the hyphal cells is without significance in itself because single spore cultures and their sub-cultures will be uniform (stable, constant strains, homotypes). On the other hand, all the nuclei may not be genetically alike; this is possible in cases (a) and (c) and it may occur in case (b) through somatic gene mutation. Further there is the condition of nuclear dissimilarity or heterokaryosis. The unlike nuclei may dissociate spontaneously, and, in spite of standardized external conditions, single spore cultures may constantly give rise to sudden new formations, saltants (unstable, labile strains, heterotypes). In such instances single spore cultures need not necessarily be pure cultures.

Such vegetative, nuclear dissociation may result either by pure accident as, for instance, by the segregation of dissimilar nuclei at the point of branching of a hypha or be favoured by a certain incompatibility between the unlike nuclei. Hence the marked tendency towards dissociation, i.e. towards instability, in certain clones.

Spontaneous nuclear segregation occurs either where hyphae branch or within the spores.

Nuclear segregation is more obvious where branching occurs. It shows itself externally by the formation of sectors differing in growth or colour

and it arises suddenly in single spore cultures some distance from the centre (Fig. 138), e.g. white or light-coloured sectors in a dark colony (albinism); in the laboratory they are sometimes called 'bud mutants' (sector type). Less frequently the aberrant mycelial patches form islands scattered over the colony (island type; Fig. 137, culture on the left).

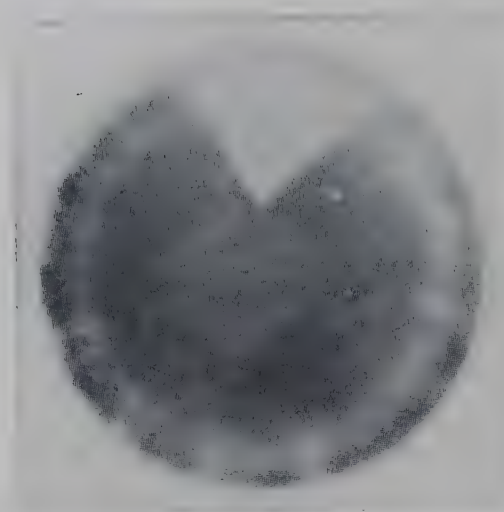


FIG. 138. Sector segregation in a single spore culture of *Fusarium*. Approx. $\times \frac{1}{3}$. (After Brown, 1926.)

Such vegetative segregation may be reproduced experimentally by cutting young hyphae into small pieces and culturing them individually. In this way Das Gupta (1934) separated a single spore culture of *Diaporthe perniciosa* (bark canker and fruit rot of

stone fruits) into three strains differing in growth and branching; the black, lined, and dotted portions in Fig. 139 indicate the original position of the different hyphal fragments.

As regards spore production on heterokaryotic hyphae, there are three possibilities: (1) unicellular spores with one nucleus; (2) unicellular spores with several nuclei; and (3) multicellular spores.

1. Unicellular uninucleate spores. If the conidia arising on a heterokaryotic mycelium are uninucleate, then one and the same conidiophore may bear dissimilar conidia. The hyphal cells of *Aspergillus niger*, for example, are multinucleate but the conidia on the other hand are normally uninucleate. Hence, in heterokaryotic clones the abstraction of conidia continually repeats the dissociation of the greater number of the nuclei; e.g. the conidia of one such conidiophore gave rise in single spore cultures to cinnamon, brown, black, and sector-forming colonies (Gossop and Yuill, 1940). One cannot therefore depend upon obtaining uniform cultures by starting with a number of spores from a single conidiophore but only by starting with a single uninucleate conidium.

Similar dissociation was observed by Das Gupta (1930) in the pycnidia of *Cytosporina ludibunda*, which causes a rot of apples; sometimes the pycnosporos from one pycnidium of this fungus differ in character.

2. Unicellular multinucleate spores. The classical examples of nuclear dissociation in single-celled, multinucleate spores are the heterothallic *Mucor-Rhizopus* spp., in which chance cleavage of the sporangial contents gives rise to sporangiospores with $+$ and $-$ nuclei or occasionally to some with only $+$ or only $-$ nuclei. A parallel from parasitology is afforded by the heterokaryotic strains of *Botrytis cinerea*. Single conidia of this fungus contain about six to eighteen nuclei, and in single spore cultures the characters cited on page 189 continually show sudden changes (Brierley, 1931). In this case, therefore, a culture that is pure arises only by chance.

3. Multicellular spores. In certain cases multicellular conidia of the genera *Fusarium* (Leonian, 1929; Ullstrup, 1935; Wellman and Blaisdell,

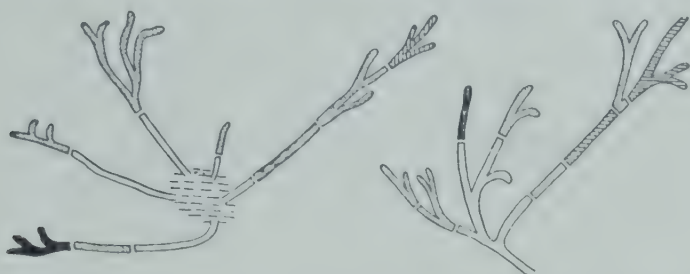


FIG. 139. Original position of the different hyphal fragments in the heterokaryotic mycelium of *Diaporthe pernicioso*. (After Das Gupta, 1934.)

1941), *Helminthosporium* (Christensen, 1926; Mitra, 1930; Fig. 141), *Clasterosporium*, &c., may arise on heterokaryotic mycelium. Where this happens the question arises whether they were originally abstricted as one-celled, uninucleate bodies which later became septate, in which case they would contain sister nuclei, or whether foreign nuclei gained entrance into them (Fig. 140). In the latter event the problem resembles that presented by the hyphal fragmentation of *Diaporthe pernicioso* as described on page 194. Single spore isolations give rise to really pure cultures only when all the cells of the conidium in question happen to contain genetically similar nuclei; otherwise, a mixture of different clones will arise from one single spore.

In such cases of nuclear dissociation it has been proved that the parasitic as well as the morphological characteristics may segregate as, for instance, in *Ophiobolus Miyabeanus* (syn. *Helminthosporium oryzae*), which causes a leaf and foot rot of rice (Hiroe, 1937). In a single spore culture of this fungus showing the island type of saltation, 16.3% of the saltants were as virulent as the parent culture, 11.3% were more virulent, and 72.5% were less virulent. In *Cytosporina ludibunda* (p. 194) some of the saltants were more and others less virulent than the original strain (Das Gupta, 1933); here the most virulent saltant permeated the host tissues fifty times as quickly as the weakest sister culture.

In addition to the spontaneous segregation so far dealt with nuclear dissociation may, to a certain extent, be induced experimentally. In this

way it is possible to isolate heterokaryotic clones and recognize their true nature, since, apart from experimental treatment, their nuclei remain



FIG. 140. The formation and abstriction of a multisepate, multinucleate conidium of *Helminthosporium gramineum*. Approx. $\times 1,000$. (After Graham, 1935.)



FIG. 141. A germinating multisepate conidium of *Helminthosporium avenae* (leaf stripe of oats). Two young hyphae have anastomosed with each other. $\times 280$. (After Dennis, 1933.)

in a condition of stable equilibrium and, therefore, rarely or never segregate spontaneously.

Among physical agents, X-rays and ultra-violet rays do not, in the majority

of cases studied, appreciably increase the tendency to nuclear dissociation (Das Gupta, 1936) although, by analogy with mutation in flowering plants, more striking effects had been anticipated. However, certain media, e.g. Richard's culture solution, high temperatures, and poisons do increase the tendency to dissociation. A rise in temperature to over 28° C. stimulates saltation in the above-mentioned *Ophiobolus Miyabeanus* with an optimum, depending on the medium, at 32° C. and a maximum at 36° C. Much the same is true for *Helminthosporium sativum* which causes a root and leaf disease of wheat, barley, &c.; most of the saltants occur at 30° C. and none below 15° C. (Mitra, 1931). In *Botrytis cinerea* it is sufficient to expose the conidia for 2 minutes to a temperature above 48° C. to release abundant saltants (Barnes, 1930).

Among chemicals, 0.01% potassium bichromate or 0.05% zinc sulphate increase saltation whereas 0.01% mercuric chloride or 0.02% copper sulphate are without effect. The possibility of favouring saltation by means of weak poisons may lead to interesting problems in the chemical control of plant diseases.

2. Changes in Parasitic Adaptation due to Vegetative Nuclear Association

The mixochimaeras of Burgeff (1915) are the classical examples of experimentally induced vegetative nuclear associations in fungi. Using micro-surgical technique he transferred plasma and nuclei of the heterokaryotic *Phycomyces nitens* forma *piloboloides* to young sporangia of the homokaryotic typical *P. nitens*. The sporangia which thus contained different kinds of nuclei were reared to maturity and the distribution of the nuclei in the spores was studied.

Similar somatic associations of nuclei occur spontaneously in the hyphal fusions of numerous plant pathogenic fungi; thus it has long been known that in some Ascomycetes and Fungi Imperfecti the hyphae anastomose vegetatively owing, it would seem, mainly to nutritional conditions. A special aspect of the problem was first indicated by the discovery that (i) the nuclei of one partner sometimes migrate into the other (Figs. 142 and 143) spontaneously bringing about heterokaryosis, and (ii) that in a large number of fungi the anastomosing strains fall into two groups which are fertile only with each other, i.e. fuse with each other by anastomosis (bipolarity, dual phenomenon). It is probable, therefore, that this is the last trace of a vanishing deuterogamous sexuality (Gäumann, 1926).

In some cases, e.g. in *Ascochyta pisi* (leaf and pod spot of pea, &c.), *Botrytis allii* (neck rot of onion), *B. cinerea* (p. 179), and *Verticillium albo-atrum* (p. 179), the vegetative nuclear association is without significance (e.g. Hansen, 1938), possibly because the fungi are completely homokaryotic or because the association leads only to a recombination of the nuclei already present and not to anything really new. This group corresponds *mutatis mutandis* to the autoecious rusts in which the heterokaryotic (i.e. dikaryotic) hyphae possess the same nutritional requirements as their parent hyphae.

In other cases vegetative anastomosis leads to genuinely new structures which are not intermediate and not, therefore, a mere summation of the parents; they correspond *mutatis mutandis* to the heteroecious Uredineae. Thus, in the case of *Aspergillus niger* (see p. 194), when a cinnamon and a brown strain anastomose with each other, black conidiophores which are new structures occur among the cinnamon and brown conidiophores. Further, Hansen and Smith (1935) produced anastomoses between single spore cultures of *Botrytis allii* which has smoke-grey mycelium and ellipsoidal conidia and *B. ricini* which has yellowish-brown mycelium and

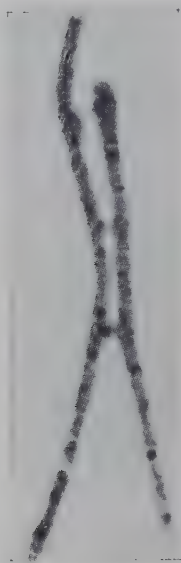


FIG. 142.



FIG. 143.

FIG. 142. Anastomoses between hyphae of two strains of *Botrytis cinerea*. One of the nuclei is about to enter the lower hyphal junction. Approx. $\times 700$. (After Hansen and Smith, 1932.)

FIG. 143. Hyphal fusions in *Hysteroglyphium fraxini*, the cause of a die back of ash. Left: bridging hypha with a migrating nucleus. $\times 2,400$. (After Zogg, 1943.)

spherical conidia. Of the resulting newly formed conidia about three-quarters resembled the two parents, while a quarter were morphologically intermediate or completely new, differing so much from the parents that an impartial observer would have described them as forming a new species.

Vegetative anastomosis can, therefore, result in the production of genuinely new structures. To explain this it is not necessary to assume that nuclear fusion has occurred, since heterokaryosis may lead to new morphological and physiological characteristics even if the nuclei remain morphologically unaffected. The rusts with alternating hosts exemplify this. In them new morphological features following heterokaryosis (dikaryon phase) are the new spore forms (aecidio-, uredo-, and teleutospores) and eventually the clamp connexions which arise on the hyphae; the new physiological result of heterokaryosis is the host change.

It is possible that the novel structures resulting from vegetative association of nuclei are instances of vegetative hybridization, i.e. hybridization by an association of independent nuclei and not by the association or recombina-

tion of their chromosomes. Experimental evidence to this effect was provided by investigations on *Neurospora tetrasperma*, one of the Sordariaceae. Under certain conditions ordinary vegetative cells in the mycelium may fuse and male nuclei pass into female hyphae. The resulting hyphae with their mixed nuclei continue to grow as if nothing had happened, and only later form ascogonia or similar reproductive primordia in which the actual pairing of nuclei and production of ascogenous hyphae take place. Before the actual dikaryon phase a developmental stage is passed through, during which the male and female nuclei exist side by side in a loose vegetative relationship in the cells of the female hyphae. These cells are therefore heterokaryotic, as in cases of vegetative anastomosis, but with the difference that the two nuclear partners come from parents of definitely opposite sex and later combine to produce paired nuclei and thereby introduce the dikaryon phase with ascogenous hyphae (Gäumann, 1940).

How do these mycelia whose heterokaryosis is sexually conditioned compare in their behaviour with the corresponding parental single spore mycelia and with mycelia which are heterokaryotic as a result of the dual phenomenon but without any actual sexual polarity? Dodge (1942) apposed three strains in turn and allowed their hyphae to anastomose, race C_4 with flesh-coloured conidia and fluffy aerial mycelium, race C_8 with yellowish-orange conidia and meagre aerial hyphae, and an X-ray-produced dwarf race, Dwarf 16, which only occasionally formed a few *Monilia*-like conidia.

Race Dwarf 16 grows very slowly, about 2 cm. per week, and is of sex a . When this is brought together with race C_4 , which is of the opposite sex A , sexual polarity results in the formation of a heterokaryotic mycelium which grows two or three times as quickly as C_4 , itself a rapid grower, and forms abundant conidia. This heterokaryotic hybrid is distinct from its parents in its particularly free growth.

If race Dwarf 16 be put with race C_8 which is of similar sex (both sex a) the heterokaryotic mycelium resulting from vegetative anastomosis is again marked by strong growth and abundant conidial formation, i.e. by the same features as the sexually produced heterokaryotic mycelium.

The greater vitality of the sexually developed heterokaryotic mycelium Dwarf $16 \times C_4$ depends not so much on particular sexual conditions but probably on the vitamin or growth substances produced by strains C_4 and C_8 , which, when brought together with that of Dwarf 16, result in an optimum for growth and conidial production. It does not matter, therefore, whether heterokaryosis comes about sexually (Dwarf $16 \cdot C_4$) or only by vegetative anastomosis (Dwarf $16 \times C_8$).

The vegetative nuclear dissociation and nuclear association dealt with under subsections 1 and 2 present, next to true hybridization (to be considered immediately), the basic problems of race formation in some parasitic fungi, and at the same time the chief cause of the segregation of morphological species into biological micro-forms. Its significance for practical phytopathology is obvious. Thus, the one barley leaf may bear two different races of *Helminthosporium gramineum*; if these happen to be

inter-fertile, they will anastomose and inaugurate a chain of new nuclear combinations, some of which will remain stable while others will segregate further by saltation, and this process may be repeated indefinitely. In this way the number of races increases endlessly year by year.

3. *Changes in Parasitic Adaptation due to Sexual Nuclear Association (Nuclear Phase Change)*

In the higher Ascomycetes and Basidiomycetes somatogamous fusion of opposite sexes (plasmogamy) does not lead at once to fusion of opposite



FIG. 144. Maize stem deformed by boils of maize smut, *Ustilago zeae*. Approx. $\times \frac{1}{4}$. Original photo. by R. Maag.

nuclei (karyogamy), but only to a nuclear association. It thus initiates a special form of heterokaryosis, the paired nuclei phase (dikaryophase) which, however, may last through the greater part of the life of the micro-organism. This is, therefore, a specifically mycological problem, which can be solved only by a thorough study of the life histories of the fungi concerned (Gäumann, 1926 and 1940).

The cells of the dikaryotic phase of the life-cycle contain potential pairs of nuclei, whose components derive from male and female parents. The contrasting nuclei, however, are and remain morphologically independent, just as ultimately the chromosomes, derived from the two parents, maintain their individuality after nuclear fusion.

Physiologically, however, they constitute a unit. Their divisions generally synchronize, i.e. they are conjugate nuclei, and they impress new characteristics upon their hyphae in exactly the same way as if the sexual process had already been completed. Among these physiological changes nutrition is important in relation to the subject under discussion because

it can effect alterations in pathogenic characteristics. The smut and rust fungi make this clear.

In *Ustilago nuda* (loose smut of barley, p. 108), one sex of the haplophase, the —mycelium, is autotrophic in regard to growth substances and grows well on, for example, potato agar, whereas the + mycelia will not develop on this medium; the latter are heterotrophic in regard to growth substances and require for their development a substance which is still unknown. The dikaryon stage is also heterotrophic in this respect and is certainly dependent on pyrimidine, which has an effect similar to that of a molecule of aneurin whose other component, thiazole, is ineffective (Thren, 1941).

The sexually conditioned heterokaryosis produces in this fungus new potentialities and responses, which cannot be interpreted merely as a summation of parental characters. In the same way, the parasitic adaptation of the two haploid sexes and of the dikaryophyte may be different; this is illustrated by another example, namely, smut of maize.

Haploid strains of *Ustilago zeae* (maize smut, Fig. 144) are as a rule unable to produce infection, and live as saprophytes; only in the paired nuclear phase do they become able to attack maize in the normal way. The nuclear association, initiated by somatogamous fusion, constitutes therefore the pre-condition for the transition from the saprophytic to the parasitic mode of life, i.e. for a change in aggressiveness: this first establishes the parasitic capacities which derive from the two parents. In the smut fungi of the maize smut type the change from the saprophytic to the parasitic way of life is dependent on the meeting of two heteropolar partners: a strain becomes able to infect maize only after it has fused with a partner of opposite sex (Table XXVII).

TABLE XXVII

Infection incidence, on Golden Bantam maize, of combinations of various strains (represented by letters of the alphabet) of Ustilago zeae (maize smut)
(After Christensen, 1929)

Strain	Combined with strain				
	A	E	F	G	H
A	—	—	+	+	+
B	—				
C	—				
D	—				

— = no infection; ± = slight infection; + = heavy infection

On account of the segregation of *Ustilago zeae* into an endless number of biotypes varying in aggressiveness and host selectivity (p. 187) it is rare for the + and — individuals of the same biotype to meet and fuse on a host plant. Most often fusions take place between + and — individuals of different biotypes resulting in the most diverse nuclear associations. Hence the spores of a single boil of maize smut generally form a very mixed population.

Conditions are probably similar in the haploid and diploid phases of certain Taphrinaceae (Gäumann, 1940).

In the rust fungi (Uredineae) sexual nuclear association has led to a very complex state of affairs; this is hardly surprising because the rusts probably date back to the Carboniferous Period.

In the first place there is variation in the sexual differentiation which conditions the sexual nuclear association. One section of the rusts is homothallic (monoecious) and here a single basidiospore can of itself inaugurate the whole life-cycle. Another section is heterothallic (dioecious) and, in this case (see Table XXVII), a mixed infection by two basidiospores of opposite sex is required for successful infection.

The rust fungi also differ amongst themselves with regard to the results of the sexual association of nuclei. About one-half are autoecious (p. 82); the sexual pairing of nuclei does not evoke any new parasitological qualities, the two nuclei being apparently of equal value in this respect. Hence, for example, in bean rust (*Uromyces appendiculatus*) and in flax rust (*Melampsora lini*) the infection chain is homogeneous but discontinuous (p. 81). It is homogeneous because, as autoecious rusts, they pass only from individuals of their host group to others of the same group; discontinuous, because they overwinter, when there are no host plants, in the form of resting spores (teleutospores). On the germination of the latter the continuous phase of the infection chain starts again; in this phase, however, no fewer than three different spore forms are produced, pycnidia, aecidia, and uredospores.

The other half, which comprises the species of economic importance, is heteroecious. Here the sexual nuclear pairing (as described on p. 84 in relation to the epidemiology of black rust) evokes fresh nutritional demands which can only be satisfied by an alternate host plant. The two nuclei brought together in plasmogamy are evidently unlike in their parasitological qualities, so that the sexual nuclear association ushers in a heterokaryotic condition with functional differences, resembling those of the partners in *Neurospora* (p. 199) which have different hormone relationships. The infection chains of these heteroecious rusts are, therefore, heterogeneous and discontinuous (p. 81).

For reasons as yet unknown alternation of hosts in the rust fungi never takes place within the same family or within the same order but always between two widely separated orders (Table XXVIII); the alternate host is generally a weed of the same plant association. In certain incompletely known rusts theoretical considerations (Fischer and Gäumann, 1929, p. 178, &c.) may lead to surmise regarding the alternate hosts. As a working hypothesis, the author would look for the aecidial host of yellow rust (*Puccinia glumarum*) among the Liliiflorae; but such predictions in the cereal rusts, which originate from the Orient and therefore from another floristic area, are always unsafe.

In order to indicate the alternating hosts in the specific epithets and thereby to aid memory, Klebahn proposed hyphenating the names of the

aecidial and teleutospore hosts (or vice versa). Thus, *Puccinia caricis-montanae* would be a *Puccinia* that formed its teleutospores on *Carex* spp. and its aecidia on *Centaurea montana*, whereas *Puccinia caricis montanae* would be a *Puccinia* that lived on *Carex montana*. This proposal for rust fungi contradicts the nomenclatural rule, Article 27, which states that specific epithets composed of two words should always be joined by a hyphen, i.e. not only in heteroecious rusts when a host alternation is to be indicated. Nevertheless, Klebahn's proposal is sufficiently useful for the special identifications in Uredinology to merit official sanction when a favourable opportunity arises.

TABLE XXVIII

Alternation of host in some cereal rusts

<i>Rust species</i>	<i>Hosts of dikaryophase</i>	<i>Hosts of haplophase</i>		
		<i>Species of the genus</i>	<i>Family</i>	<i>Order</i>
<i>Puccinia graminis</i> (black rust)	Numerous Gramineae	<i>Berberis</i>	Berberidaceae	Polycarpicae
<i>Puccinia triticina</i> (brown rust of wheat)	<i>Triticum</i> spp.	<i>Thalictrum</i>	Ranunculaceae	Polycarpicae
<i>Puccinia hordei</i> (dwarf rust of barley)	<i>Hordeum</i> spp.	<i>Ornithogalum</i>	Liliaceae	Liliiflorae
<i>Puccinia dispersa</i> (brown rust of rye)	<i>Secale</i> spp.	<i>Anchusa</i> and <i>Lycopsis</i>	Boraginaceae	Tubiflorae
<i>Puccinia coronata</i> (crown rust)	Numerous Gramineae	<i>Rhamnus</i>	Rhamnaceae	Rhamnales
<i>Puccinia glumarum</i> (yellow rust)	Numerous Gramineae	Unknown		
<i>Puccinia sorghi</i> (maize rust)	<i>Zea</i> and <i>Euchlaena</i>	<i>Oxalis</i>	Oxalidaceae	Gruinales

Host alternation in rust fungi is correlated with a specific course of development and, therefore, it must fail if the latter be interrupted. One case, apomixis with homogenization of the infection chain on the aecidial host, has already been discussed (p. 85). The other case, shortening of the life-cycle, again with homogenization of the infection chain on the aecidial host, leads us back again to rust fungus theory.

In addition to those rusts with a complete life-cycle, the so-called euforms (see scheme on p. 85), there are others with a shortened cycle, i.e. they no longer produce all the spore types. Thus, the microcyclic forms possess only teleutospores, and occasionally pycnidia, and somatogamous fusion occurs in the hyphae before or during the formation of the mycelial knot which will give rise to the teleutospores. The teleutospores of those microcyclic forms which arise on the aecidial host of a euform in some cases strikingly resemble the teleutospores on the alternate host of the euform to which the aecidia belong.

An example will make this evident. The alpenrose rust (*Chrysomyxa rhododendri*) forms its pycnidia and aecidia in summer on the needles of

spruce (*Picea excelsa*). At the end of July, or in August or September at higher altitudes, the aecidiospores are mature. Their germ tubes enter the stomata of adult alpenrose leaves (alternation of host) and there develop a mycelium with paired nuclei which forms uredospores that spread the fungus from one alpenrose to another. The fungus then overwinters, and early in the following summer produces sori of teleutospores. The teleutospores germinate before or during the flowering period of the alpenrose (June, July) and without a resting stage, i.e. directly on reaching maturity, which is a characteristic feature of the so-called leptofoms; the basidiospores reach the young spruce needles which are just breaking bud at this time. Their germ tubes penetrate the epidermis (alternation of host). Ten days after infection the first pycnidia appear.

Beside this alpine heteroecious euform, there occurs on spruce at moderate altitudes an autoecious microcyclic species, the pathogen of the so-called needle rust (*Chrysomyxa abietis*) which forms teleutospores only. These are formed on the needles in the autumn, mature in May and, like the leptofom, are capable of immediate germination. The basidiospores directly infect other young spruce needles. The teleutospores of the autoecious, microcyclic *C. abietis* on *Picea excelsa* agree so completely in the structure of their sori, in their characteristic form and in their biological behaviour (leptofom) with the teleutospores of the heteroecious, macrocyclic *C. rhododendri* on alpenrose, that it has long been suggested that they have arisen as a secondary, derivative species from the primary *C. rhododendri*, through a condensation of the life-cycle so that the teleutospores are formed on the spruce immediately after somatogamous fusion.

Experimental evidence that such autoecious rusts can be formed from heteroecious species by a condensation of the life-cycle was first brought forward by Newton and Johnson (1937) and Johnson and Newton (1938) for black rust. They crossed biotypes 9 and 36 of *Puccinia graminis tritici*; the F_1 and F_2 generations behaved normally. Also in F_3 the basidiospores produced the usual pycnidial mycelium on *Berberis vulgaris*; no aecidia followed the pycnidia but 44 days after the infection and in direct connexion with the pycnidia, sori of uredospores and later of teleutospores appeared which, *lege artis*, should have arisen on wheat. There is still no cytological explanation of this anomaly but this is the first instance of the artificial production, or cultivation, of an autoecious reduced form with pycnidia, uredospores, and teleutospores (a so-called brachyform) from a heteroecious euform and it is thus an entirely new species. If it were highly pathogenic it could give rise to a new epidemic on *Berberis*.

4. Changes in Parasitic Adaptation due to Hybridization

Novelties similar to those resulting from somatic nuclear association and dissociation may arise in the sexual reproduction of fungi and persist because of their chromosomal basis. Doubtless in nature spontaneous hybridizations continually occur between $+-$ and $--$ individuals of different races, which by way of ascospores, basidiospores, &c., give rise

to new hybrid biotypes similar to those produced directly through somatic hyphal fusions.

In general the biotypes do not survive sexual reproduction but disappear if the same genetic combination does not by chance recur. The segregation of biotypes illustrated on page 189 is, consequently, only possible in those pathogens which reproduce asexually and vegetatively by conidia or uredospores and so by-pass or exclude the sexual new combination of genetic material. If asexual propagative spores are lacking and every reproduction is a sexual one as in the pear rust, *Gymnosporangium Sabinae* (p. 86; in the aecidia, plasmogamy, and in the teleutospores, karyogamy), then biotypes cannot develop at all.

The more finely adjusted the host selectivity of the pathogen the more strikingly will it be affected by genetic change. Three examples, the fungus of apple scab, a rust, and a smut will show this.

At times hardly two isolations of the pathogen of apple scab (*Endostigme inaequalis*) will belong to the same race (p. 187). *Endostigme inaequalis* is heterothallic and practically every fusion is a hybrid fusion leading to recombination of genetic material and, therefore, to the forming of new biotypes. Keitt and Palmiter (1938) have shown that segregation occurs at the reduction division. They made single spore cultures from the eight ascospores of one ascus and found that these differed in their morphological and cultural features (see p. 187) and also in their host selectivity in relation to four test varieties (Table XXIX; it is remarkable that the aggressiveness for the variety Fameuse is not affected by the segregation). Further, as *E. inaequalis* is heterothallic, Table XXIX indicates a difference in aggressiveness between the two sexes: one sex is highly virulent for the first two test varieties, the other almost non-virulent, while their parasitic activity in relation to the last two test varieties is directly reversed.

TABLE XXIX

Host choice by single spore cultures from the eight ascospores of one ascus of Endostigme inaequalis. (After Keitt and Palmiter, 1938)

Test variety	Isolation no.							
	1	2	3	4	5	6	7	8
Yellow transparent . . .	+	—	—	+	—	—		+
McIntosh	+	—	—	+	—	—	+	+
Missouri Pippin	±	+	+	±	+	+	±	±
Fameuse	+	+	+	+	+	+	+	+

+ = Severe infection with abundant conidial production.

± = Slight infection with slight conidial production.

— = Sub-infection or no attack.

Black rust of cereals (*Puccinia graminis*, p. 84) will be taken as an example of a hybridizing rust fungus. Let us consider a barberry bush (*Berberis vulgaris*) situated in the middle of a cereal field infected by black rust.

Every spring the basidiospores of different biological species and biotypes infect the young barberry leaves and develop haploid, pycnidial-forming mycelia which initiate the sexual processes. In addition to legitimate fusions between members of the same biological species or race, different biological species and races meeting by chance on their collective host, barberry, also fuse with each other (hybridization) and produce heterokaryotic hybrid aecidiospores. Some of these aecidiospores will be distinguished by an aberrant host range, which may be either more restricted or more extensive.

An example of restricted host range was provided by Johnson *et al.* (1932) who crossed biotype 95 of black rust of wheat (*Puccinia graminis tritici*) with a biotype of black rust of rye (*P.g. secalis*) on *Berberis vulgaris*. They obtained, among others, a completely new race of rust, *P.g. tritici* biotype 111, with such insignificant parasitological features that its inclusion in the *tritici* group seemed almost arbitrary. Its power to infect either wheat or rye is much reduced, but it is intermediate between that of its parent strains: of the twelve test varieties of wheat susceptible to the *tritici* parent, strain 111 infected only one and that weakly and of the test varieties of rye susceptible to the *secalis* parent strain, it attacked a few weakly and two not at all. As regards barley, a subsidiary host, its aggressiveness remained as slight as in the two parent strains. In this example, therefore, hybridization effected a marked restriction of host selectivity.

In certain other cases the consequence of hybridization is a widening of host range, and this is of practical importance. Thus Stakman *et al.* (1930) crossed black rust of wheat (*P.g. tritici*) with black rust of wind bent grass (*P.g. agrostidis*), and, from a single aecidium, obtained no fewer than eight biotypes, some of them new, and one of them even able to infect a particular variety of emmer wheat (*Triticum dicoccum*); had this occurred in the open the first conditions would have been created for a new epidemic on this host.

Rules of inheritance which allow of the prediction of host ranges and pathogenic characteristics of hybrids do not yet exist. The genetic processes are so complicated that Johnson and Newton (1940) suggest, on the basis of their experimental crossings of black rust of oat (*P.g. avenae*), that a lack of congruence between the parents is involved with unequal cytoplasmic inheritance, since each hybrid aecidiospore receives only the nucleus from the male parent and both nucleus and cytoplasm from the female parent.

In every case, therefore, barberry bushes in the middle of a cereal field infected with black rust are continually creating new possibilities of hybridization and thus form a permanent source of novel hybrid rust biotypes with new host ranges. This has been proved statistically by Stakman *et al.* (1934). In 94 aecidiospore isolations of *P.g. tritici*, 26 biotypes were found (of which 6 were new), i.e. 1 biotype per 4 aecidiospores. This is to be compared with 8,000 uredospore isolations made at random all over the country, of which 100 isolations gave one biotype,

i.e. 25 times fewer than for the aecidiospores. Hence the production of new rust strains with new host spectra never ceases on the barberry.

The destruction of the barberry (or other alternate hosts for other rust diseases) in cereal-producing areas not only interrupts the infection chain in its heterogeneous phase (p. 145), but above all deprives the pathogen of the possibility of bringing new biotypes into the world through hybridization. Each new biotype is, under certain circumstances, a new pathogen and generates a new disease which may cancel out all the earlier painstaking work of plant breeders.

Hybridization experiments similar to those with black rust were made with smut fungi. On *Avena* spp. there occur two morphologically indistinguishable smuts, *Ustilago avenae* (loose smut of oats) on cultivated *Avena* spp. and the smut *U. perennans* on *Arrhenatherum elatius* (tall oat grass). Each year the germ tubes of the first penetrate anew the coleoptiles of the seedlings, whereas the mycelium of the latter fungus perennates in the rhizome. The sori of the first smut, as its name indicates, are generally naked and powdery although exceptionally they may be enclosed by the glumes of the grain; the sori of the second smut are, on the other hand, regularly surrounded by the partially destroyed empty glumes.

What happens when the two fungi are crossed? In the segregations, according to Holton (1941) and Fischer and Holton (1941), naked powdery sori are dominant to covered compact sori, though all four combinations may occur, naked-powdery, naked-compact, covered-powdery, and covered-compact. *Arrhenatherum elatius* is not susceptible to *U. avenae* nor to the hybrid *U. avenae* × *U. perennans*; in contrast, *Avena sativa* is susceptible to *U. perennans*. In hybridization, resistance to *U. perennans* is dominant. Incidentally, these experiments indicate that *U. perennans* must be withdrawn as a distinct species and must be considered only as a *forma specialis* of *U. avenae*.

5. Changes in Parasitic Adaptation due to Mutation

True mutations result from spontaneous changes in the genome, i.e. the hereditary material carried in the chromosomes, or their homologues among the bacteria. This distinguishes them, on the one hand, from true saltations which derive from new combinations of nuclei which do not, themselves, undergo genetic change, and on the other hand, from hybrids in which the new combinations of hereditary material result from a sexual process.

In multinucleate bodies it is naturally difficult to decide whether a suddenly occurring change is due to a spontaneous nuclear association or dissociation, or whether it was originally based on a true mutation. For this reason examples are chosen in which the suddenly changing cultures originate from material which, genetically, is free from criticism. When mutations appear in such cultures they are true somatic gene mutations,

brought about either by non-disjunction (failure of the chromosomes to separate at meiosis) or by similar processes.

In plant pathogenic fungi, mutation either changes aggressiveness quantitatively (e.g. *Ustilago zeae*) or host selectivity qualitatively (e.g. *Puccinia glumarum*).

Ustilago zeae (Fig. 144) segregates into an almost unlimited number of haploid strains, distinguished from one another by every possible kind

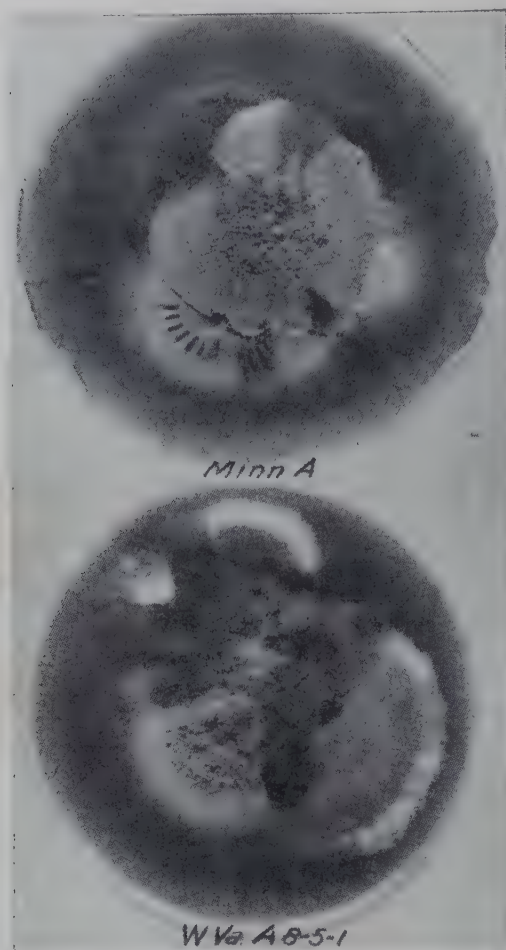


FIG. 145. Sector and island type mutations in two single spore cultures of *Ustilago zeae*. Approx. $\times \frac{1}{2}$. (After Stakman *et al.*, 1929.)

of physiological and cultural feature. Stakman *et al.* (1929) examined about 200 single spore cultures all derived from a single uninucleate basidiospore; the majority were genetically unstable and mutated in sector or island form (Fig. 145). The mutants differed from each other in colour, consistency, margination (Fig. 146), temperature requirements, sex determination, power of liquefying gelatine or digesting casein, reducing nitrates, &c. Certain strains mutated freely (in the course of a year one formed 220 distinct mutants, another 162), others only seldom. The aggressiveness of these strains generally cannot be tested directly, because being haploid they are not as a rule able to cause infection (p. 201). By the use of combinations it can be shown that many of them have lost their aggressiveness through the mutation and can no longer produce infection even in the dikaryophase, whereas others are more highly pathogenic than the original strain.

Similar unquestionable mutations were described by Dickinson (1932), e.g. in *Helminthosporium pedicellatum* which causes a foot rot of wheat, in *Fusarium fructigenum* which causes a bud and fruit rot of apples, and in *F. vasinfectum* which causes wilting of trees in North America. The septate conidia of these fungi are originally abstricted as one-celled uninucleate bodies, so that true somatic mutation must have occurred. Further examples are given by Das Gupta (1930) for the ascospores of *Diaporthe pernicioso* (p. 194), and by Walter (1937) for the pathogen of elm disease *Ophiostoma ulmi* (syn. *Ceratostomella ulmi* = *Graphium ulmi*), &c.

In all these cases the sudden changes relate to various cultural characters or to aggressiveness and pathogenicity. The host range may be extended

by mutation as, for example, in the yellow rust race *Emersleben* (Gassner and Straib, 1932).

Biotype 9 of *Puccinia glumarum* which had been isolated originally from Heines Kolben and v. Rümkers club wheats from Emersleben attacked



FIG. 146. The single spore culture of *Ustilago zeae* shown in the upper half of Fig. 145 (in Fig. 146 on the lower right, 'Par') with seven mutants which differ from the parent strain in colour, texture, and margination. Approx. $\times \frac{1}{3}$. (After Stakman *et al.*, 1929.)

many wheat varieties only slightly, and this was especially the case with many club wheats, which are very susceptible to other forms of yellow rust. On the other hand, Heines Kolben and v. Rümkers early spring club wheats, which are both highly resistant to other forms of yellow rust, can become severely infected.

The single spore culture 126 on the very susceptible test variety, Heines Kolben wheat, gave a severe infection with numerous uredo-pustules

without a true necrotic halo (infection type 4, Fig. 147 *a*). On the highly resistant test variety Strubes club wheat, at an air temperature of 10–20° C.

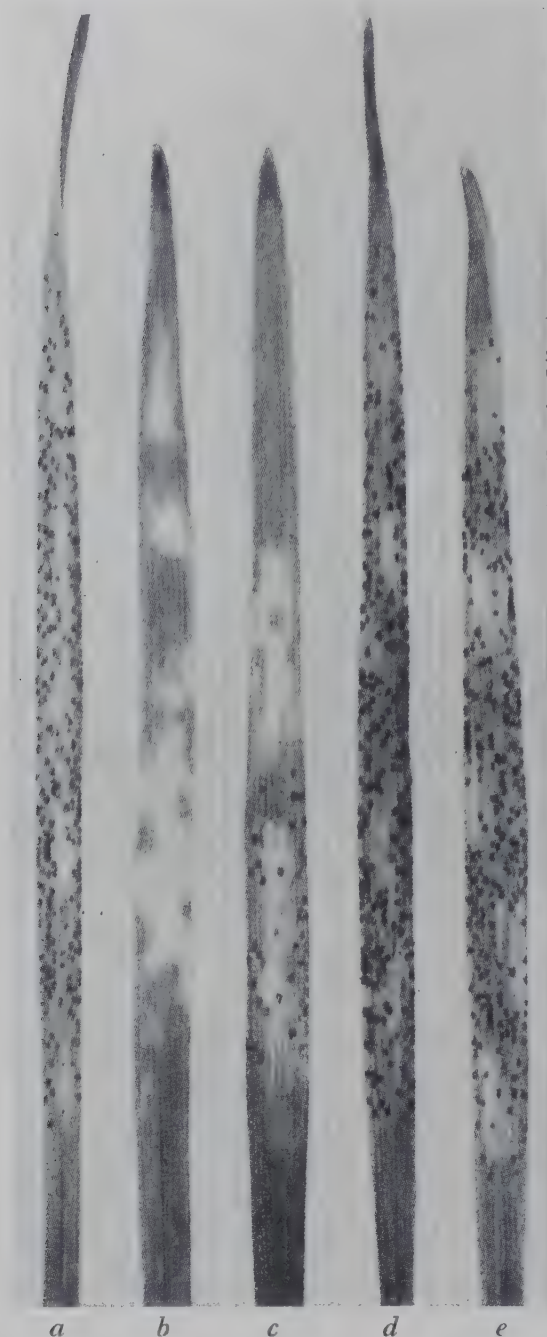


FIG. 147. Change of host range in a race of yellow rust by mutation. *a* clinical picture produced by single spore culture 126 on the susceptible Heines Kolben wheat; *b* on the resistant Strubes Dickkopf wheat; *c* appearance of the mutant on Strubes Dickkopf wheat; *d* clinical picture of the mutant on Heines Kolben wheat and *e* on Strubes Dickkopf wheat. Approx. nat. size.

(After Gassner and Straib, 1932.)

only sub-infection was produced, i.e. necrotic flecks without uredopustules (infection type 0, Fig. 147 *b*). It was propagated on Heines Kolben wheat from which 10–20 plants of Strubes club wheat were inoculated each time as experimental and control plants. During the first four generations no peculiarity appeared. In the fifth generation two individuals of the resistant Strubes club wheat each bore a group, about 2–3 cm. long, of normal, large uredopustules, so that the clinical picture in these areas approximated to infection type 4 (Fig. 147 *c*), but outside these areas the former infection type 0 remained.

Single spore inoculation of this new material produced infection type 4 on both Strubes and Heines club wheats (Fig. 147 *d* and *e*). In this case we are dealing with a new mutant which differs from the original strain in its extended host range. Within the group of the test varieties already mentioned it can also attack Strubes club wheat which the original strain was unable to do. The same mutant was subsequently formed over thirty times from the single spore culture 126, and the mutation rate is of the order 1:1–200,000.

When compared with the wheat varieties attacked by biotype 9, the new mutant is equally aggressive; the mutation has produced no loss of capacity. Some wheat varieties which were resistant to biotype 9 remain resistant to the mutant; so

far as these varieties are concerned the host range has not been affected by the mutation. The majority of wheat varieties which are resistant to biotype 9 are, on the other hand, heavily attacked by the mutant. On

account of the mutation the host range of the mutant is not only enlarged to include Strubes club wheat but also nearly all those wheat varieties which are resistant to biotype 9; thus, among these formerly rust resistant varieties of wheat, conditions are present for the outbreak of a new epidemic.

As an example of mutation in bacteria, *Bacterium Stewartii*, the cause of the North American maize wilt (p. 169) may be cited. Lincoln (1940) injected single cell cultures into maize plants and made isolations from the diseased tissues. With certain strains he obtained mutations, e.g. in regard to the colour of the colony (white, pale, or dark yellow) or in aggressiveness. The mutation rate (the proportion of mutants to the total number of colonies plated out) varied from 1:20,000 to 1:800,000, a similar range to that found for gene mutation in higher plants. The occurrence of back-mutations (chiefly from dark yellow to light yellow and back, and from dark yellow to white but never from white to yellow) leads one to suppose that some of these mutations cannot be explained as loss mutations. According to its resistance the host selects the strains of higher or lower virulence (p. 169) and, therefore, such selection leads, in the case of strain mutations occurring in the host tissues, to a radical change in the pathogenicity of an originally pure and uniform bacterial strain.

In the plant pathogenic viruses, as in fungi and bacteria, mutation may be common or rare but the mutation rate cannot be estimated numerically because the ratio of mutants to the remaining virus particles cannot be calculated. The virus mutations which have been established so far affect mainly the group of factors concerned with aggressiveness, particularly the intensity and quality of the symptoms produced.

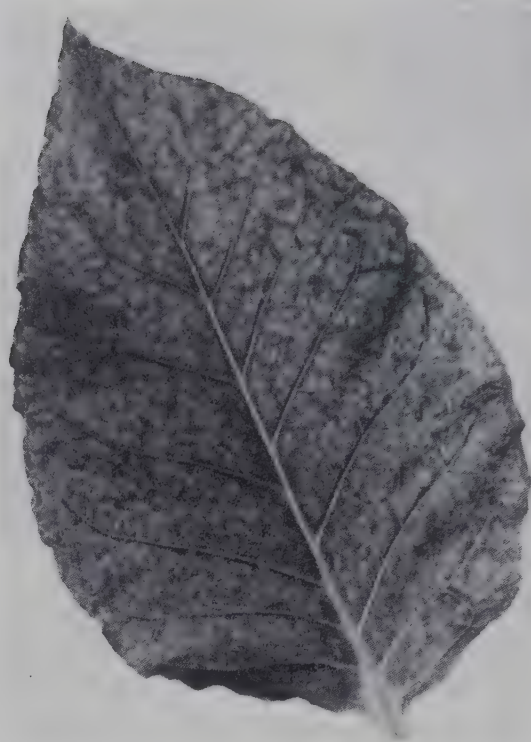
Expressed sap from tobacco leaves infected with the strain Cs 35 of the relatively unstable potato mosaic virus X, which produces ring-like chlorotic flecks often with a tortoiseshell pattern (Fig. 148 *a*), was kept for 24 hours at a constant temperature of 50° C. When this sap was rubbed on to tobacco leaves (Köhler, 1937, 1939, and 1941) it became evident that a new mutant Cs 36 giving a much better defined tortoiseshell pattern had arisen (Fig. 148 *b*). In a subsequent year, expressed sap of strain Cs 35 was kept for 10 hours at 64° C. and two new mutants were obtained, Cs 64 1 (Fig. 148 *c*) and Cs 64 2 (Fig. 148 *d*) which resembled the particularly aggressive strain Cs 36 in the severity of the symptoms but differed from it qualitatively in the highly necrotic primary lesions. Thus, within 2 years there were produced experimentally from the one strain, Cs 35, three new mutants differing widely among themselves.

The external factors which favour the production of true mutations in fungi, bacteria, and viruses are the same as those favouring saltations.

(*a*) High temperatures. With a particular strain of *Ustilago zeae* Stakman *et al.* (1929) obtained no mutations at 8° and 15°, 1 at 20°, 8 at 26°, and no fewer than 18 at 31° C. In the case of viruses in expressed sap, temperatures near to the inactivation point (about 66° C.) are the most effective, whereas inside the host plant a far lower temperature (e.g. 35° C.) can induce mutation.



a



b



c



d

FIG. 148. Induced mutations in a strain of potato mosaic-X-virus. *a* original strain Cs 35 with its three mutants, *b* (Cs 36), *c* (Cs 64/1), and *d* (Cs 64/2), all on tobacco leaves. Approx. $\times \frac{2}{3}$. Original. Photo. E. Köhler.

(b) Poisons. For example, zinc salts (Dimock, 1936) and also in rare cases Röntgen and ultra-violet rays (Das Gupta, 1936; Rodenhiser and Maxwell, 1941).

(c) Passage through uncongenial hosts. The viruses of the potato X-group can be induced to mutate by passage through certain Chenopodiaceae and those of the Y-group by passage through *Schizanthus* (Salaman, 1938).

6. *The Influence of Nutrition on the Parasitic Adaptation of Pathogens*

Before a pathogen can absorb materials from its host it must dissolve the cell walls by enzyme action, and then withstand and subsequently overcome the resistance of the host cells. These are functions of aggressiveness. Only after a successful start has been made and the infection has 'taken' can the pathogen live upon the host.

This initial period creates difficulties of two kinds for the pathogen. Firstly, it must possess the necessary vitality for parasitic attack; the greater its vitality the greater, *ceteris paribus*, will be its aggressiveness. Secondly, it must be able to reorientate itself sufficiently quickly to the exigencies of the parasitic way of life and nutrition.

The biotrophic pathogens of group 1 (p. 57) provide their spores with sufficient reserve nutriment to feed the germ tube until infection has 'taken' and the first haustoria have developed. For them the parasitic start is, therefore, no problem.

In contrast, for most biotrophic pathogens of group 2 (p. 58) and many necrotrophic pathogens (p. 59) the maternal provision is not sufficient to last until the stabilization of the parasitic relationship. Hence, in order that the germ tube may acquire and retain the necessary vitality it must have at its disposal sources of saprophytic nourishment in addition to the maternal store. For example, if a tomato fruit be inoculated with a water suspension of sporangiospores of the pin mould, *Mucor stolonifer*, no infection is obtained, but if they are inoculated with a nutrient suspension, infection usually occurs (Nobécourt, 1928). Thus, the epidermis of the tomato can resist weakly growing hyphae of *M. stolonifer* but not vigorous hyphae. On the other hand, the tomato epidermis is always an adequate protection against *Penicillium 'glaucum'*.

Supplementary nourishment of the young germ tube is often provided by rotting plant fragments which, in one way or another, reach the organ subsequently infected; infection can start from these.

When, for example, petals in greenhouses or elm fruits in the open in wet weather fall on to the foliage leaves of primulas, pelargoniums, peas, &c., they rot there and become infected by grey mould (*Botrytis cinerea*). In this way the fungus can become vigorous enough to pass over to the healthy leaf tissues which the germinating spore was not able to attack directly. Hence, the common occurrence under such a rotting petal of a brown discoloration or a perforation, due to the fungus, on an otherwise intact foliage leaf. For this reason, because the fallen corollas may lead

to *Botrytis* rot on the leaves and so to a deterioration of the product, tobacco cultivated for the outside leaves of cigars is often de-budded in the field.

In a similar way *B. cinerea* can use dead cotyledons of beans, &c., as starting-points from which to invade the healthy tissues and cause a lethal wilt disease.

The state of nutrition is decisive for parasitic success not only in the case of germinating spores but also for those pathogens which are temporarily saprophytic in the soil and thence attack their hosts.

The honey agaric (*Armillaria mellea*) cannot easily attack vigorous conifer tree trunks directly from the soil, but, if it can feed more richly on neighbouring old stumps than is possible merely in the forest soil, it can become vigorous enough to penetrate undamaged roots in the soil by means of its rhizomorphs (p. 44). Hence Armillaria disease is often found in the vicinity of old stumps.

A similar case is found amongst the timber-destroying fungi. It is only in the most exceptional cases that a hypha or the germ tube of a basidiospore of *Mauerschwamm* (*Polyporus vaporarius*), cellar fungus (*Coniophora cerebella*), or of a *Lenzites* sp. can infect sound fir beams or boards even under optimal conditions of temperature and moisture. If, however, the fungi find their way on to sound wood via rotting splinters, shavings, &c., or, as in the laboratory, in a fragment of agar, then the start is made easier and infection takes place.

This question of the start of infection is fundamental in relation to the impregnation of telegraph and other poles. Destructive fungi can rarely attack directly the undamaged heart-wood of Scots and Arolla pines and larches, hence the almost unlimited durability of the resistant sap-free heart-wood of these trees. If, however, in the mountains, an untreated trunk of one of these species is used as a telegraph pole, then the fungi initially attack the sap-wood, where they gain strength, and attack the heart-wood secondarily. Hence the aim of impregnation is to prevent the fungus from gaining a start at the place of least resistance by poisoning the sap-wood. In consequence the non-impregnated and impregnable heart-wood remains sound. Owing to the inability of these fungi to gain an initial foothold on the heart-wood, the impregnation of the sap-wood of pine and other poles is adequate for practical purposes.

Lastly, the problem of the start of the pathogen is especially important in the technique of laboratory culturing. The parasite is almost forced to live on artificial substrates and its diet is unbalanced since it is overfed with carbohydrate and nitrogen compounds. In the sudden transference on to a formerly suitable host plant, the conversion to the original parasitic mode of life cannot always be accomplished rapidly enough. Aggressiveness has diminished, and before the pathogen can be used in experimental work it must be regenerated by culturing on poor, slightly acid substrates, e.g. plant stems, &c.

Thus, the parasitic adaptation of a pathogen may differ according to the

previous culturing of the infective organism. In *Ophiobolus graminis* (p. 99), for example, it is optimal after previous culturing on straw (Table XXX), usually less so after oat and barley grains because of overfeeding, and least on soil because of starvation. The Polyporaceae which are parasitic on the trunks of living trees, e.g. *Fomes igniarius*, the false tinder fungus of apple trees, &c., and *F. fomentarius* which causes a white rot of beech trees, are more successful parasites when they have previously been grown on wood from suitable trees than if they have been cultured on bread or agar (Grosjean, 1942).

TABLE XXX

The influence of the previous nutrition of Ophiobolus graminis on the percentage of diseased wheat plants. (After White and McIntyre, 1943)

Nutrient medium	Strain no.					Control
	1	3	4	5	6	
Straw	10	74	62	89	0	0
Oat and barley grains . .	0	4	76	3	0	0
Soil	0	48	2	0	0	0

But even with culture media rich in carbohydrates variations occur in difficulty of readaptation. Thus, *Pythium de Baryanum* previously cultured on oatmeal induced only half as much root rot of *Pinus* seedlings as after culture on steamed rice. Similarly, *Gibberella Saubinetii* after previous culture on oatmeal killed only 10% of *Pinus* seedlings, whereas after culture on steamed rice, under the same external conditions, 50% were killed (Rathbun, 1925). Similar differences in aggressiveness were produced by unbalanced feeding on certain nitrogen compounds (see Table XXXI). In this experiment *Sclerotinia Libertiana* (syn. *Sclerotinia sclerotiorum*), a polyphagus parasite on most cultivated plants and vegetables, was cultured for a year on nutrient substrates the basic constituents of which were 2% saccharose, 0.5% KH_2PO_4 , 0.25% MgSO_4 , and 2% agar.

TABLE XXXI

The influence of unbalanced nutrition and various sources of nitrogen on the virulence of Sclerotinia Libertiana for tomato seedlings, other conditions remaining constant. (After Bunschoten, 1933)

Source of nitrogen	Dead seedlings
0.5% $(\text{NH}_4)_2\text{SO}_4$. . .	83.8
0.5% $(\text{NH}_4)_3\text{PO}_4$. . .	71.5
0.4% NH_4NO_3 . . .	58.3
0.5% NH_4Cl . . .	54.3
0.3% Urea . . .	53.5
1.0% KNO_3 . . .	44.7
0.5% Asparagine . . .	13.8
2.0% Peptone . . .	2.9

In sensitive organisms, pampering by domestication with consequent

lowering of pathogenicity naturally increases with the duration of laboratory culturing. Thus (Table XXXII), the incubation time for black spot of rose doubles between the first and third months of pure culture on malt agar, and successful infections decrease to almost one-tenth.

TABLE XXXII

The influence of the duration of artificial culture on the parasitic adaptation of Diplocarpon rosae (black spot of rose). Variety used in experiment: W. E. Chaplin. (After Frick, 1943.)

<i>Spore material</i>	<i>Incubation period</i>	<i>Diseased leaflets</i>
	<i>days</i>	<i>%</i>
Fresh summer conidia .	16	53.2
1-month-old pure culture .	16	50.9
3-months-old pure culture .	31	6.7

After years of unbalanced culturing in the laboratory certain micro-organisms undergo such profound disturbances in their metabolism, e.g. fatty degeneration, or so lose the habit of forming cellulase and thus of dissolving cell walls, that they degenerate, lose their aggressiveness, and become parasitically lazy and phenotypically saprophytic. *Bacterium solanacearum*, the pathogen of the tropical slime disease of tobacco, and *B. tabacum* (p. 18) entirely lose their capacity for infection only a few months after being isolated, whereas certain other micro-organisms are very tolerant in this respect and can retain their capacity for growth and infection for years, e.g. *Corticium vagum* (p. 179) and *Bacillus atrosepticus* (p. 54) for at least 30 years on neutral broth agar.

7. *Changes in Parasitic Adaptation due to the Host (the Passage Problem)*

While the previous subsection dealt with the influence of pre-infectious nutrition on the parasitic adaptation of the pathogen, the changes in the pathogen during the parasitic relationship will be considered here. In the host not only the host-specific nutrition but all the other endophytic influences are operative, including the defence reactions.

In the great majority of cases the pathogen does not undergo any measurable change in its parasitic adaptation due to the host. However, there are cases in which its parasitic adaptation is definitely increased by passage through susceptible hosts and reduced by passage through new uncongenial hosts or, conversely, weakened by susceptible hosts and strengthened by uncongenial hosts. Only these labile forms will be considered here.

The passage effect was first recognized in human and veterinary medicine. Here it can take the form of a heightening of aggressiveness which facilitates and accelerates the multiplication of the disease germs in the host organism which, in turn, leads to an increased malignity of the pathogen or a rise in its pathogenicity.

Thus, 1 c.c. of an 18-hour-old broth culture of streptococci freshly isolated from man can be tolerated by a mouse, but the virulence of such streptococci can be increased 100,000 times by successive passages through mice so that even 0.00001 c.c. is lethal. The opposite effect, reduction in virulence by passages through non-congenial hosts, plays an important role in serum production.

If rabies virus ('street' virus of Pasteur) from a dog be inoculated into the brain of a rabbit and then transferred 20–40 times from rabbit brain to rabbit brain, the incubation time decreases from about a month to a minimal value of 9 days at which it remains constant; hence Pasteur's term, '*Virus fixe*'. Its aggressiveness to rabbits has thus markedly increased as a result of passage through rabbits. At the same time its pathogenicity to rabbits is also increased; after the first transference to a rabbit only slight symptoms of the disease may appear, whereas at the end of the series of passages the virus is usually fatal.

Similar changes occur in fowl pest; by passage through ducks its virulence is increased for ducks but decreased for fowls. However, in the viruses it is difficult to prove conclusively the existence of a true alteration in virulence as there always remains the objection that the initial virus was a mixture and that passage has isolated and selectively favoured one component.

In the plant pathogens the passage problem has two aspects: (1) the effect of passage through organs deleterious to the pathogen, and (2) the effect of passage through stimulating or unsuitable hosts.

Firstly, the influence of specific organs. When a strain of *Phytophthora infestans* (blight of potato) lives or is cultivated for years, i.e. for perhaps 100 generations, upon tubers of an otherwise suitable variety, passing directly from one tuber to another, it degenerates (Orth and Lehmann, 1935). The germinability of its sporangia decreases from about 90–92% to about 10–15% and its ability to infect the haulms of the variety concerned diminishes until it can no longer attack. Although the tubers belong to an otherwise congenial variety, the pathogen degenerates through long continued overfeeding on carbohydrates and its aggressiveness is impaired. But if it is painstakingly transferred back to foliage leaves of this variety under optimal conditions for infection, the germination capacity of its sporangia increases even after the first passage (Fig. 149) from 20 to 74%, and after the third passage to 89%, whereas the controls grown on tubers remain at their low germination capacity. At the same time the fungus regains its original pathogenicity so that the clinical picture does not differ from that of parallel cultures grown permanently on potato plants.

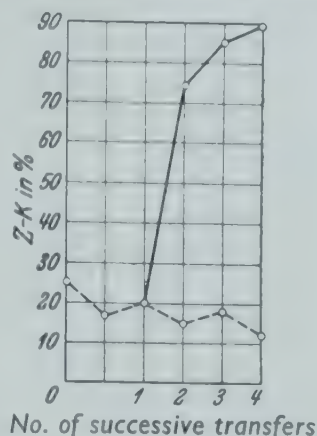


FIG. 149. The alteration in the germinability of the sporangia of *Phytophthora infestans* when continuously cultured on tubers (dotted curve) and then transferred to leaves of the same variety (continuous line). Ordinates: % germination ($z-k$ = zoospore germination). (After Orth and Lehmann, 1935.)

Continuous culture on the too favourable tissues of a congenial host plant has 'pampered' and enfeebled the pathogen and rendered it temporarily unfit for life.

Secondly, the change in the parasitic adaptation of a pathogen due to particular hosts shows four aspects: (a) increase in the virulence of a pathogen by passage through congenial hosts, (b) its increase through resistant hosts, (c) the alteration of the host range by host passage, and (d) the nature of the alteration effected by passage through hosts.

(a) Increase in the virulence of a pathogen by passage through congenial hosts and its decrease by passage through non-congenial hosts.

If the virus of curly top (*Kräuselkrankheit*) of sugar beet be transferred to nettle-leaved goosefoot (*Chenopodium murale*), to curled-leaved dock (*Rumex crispus*), or to *Suaeda Moquini*, and from these inadequate hosts, on which it only produces negligible symptoms, be transferred back again to sugar beet, it is then only slightly infectious (Table XXXIII) and evokes only a mild form of curly top (Carsner, 1925; Lackey, 1931).

TABLE XXXIII

Attenuation of the virus of curly top of sugar beet by a passage through Chenopodium murale and its regeneration by a passage through Stellaria media. (After Lackey, 1931)

<i>Condition of virus</i>	<i>Diseased sugar beets</i>	<i>Average incubation period</i>	<i>Severity of symptoms</i>
	%	days	
Original virus . .	64	9.5	Severe
Weakened virus . .	25	13.1	Very mild
Regenerated virus . .	74	9.9	Severe

The infectivity of the virus is thus greatly reduced by uncongenial passage and its pathogenicity nearly lost; one more step and infection would remain inappreciable or latent.

Passage through an unaccustomed species such as chickweed (*Stellaria media*) restores the original pathogenicity and infectivity to a remarkable extent (Table XXXIII).

If a given strain of *Phytophthora infestans* be inoculated into a tuber of a resistant potato variety and transferred from tuber to tuber, it becomes attenuated in the course of a few weeks and dies out after five to eleven generations (K. O. Müller, 1936). Conversely, if the process has not gone too far, the strain regains its original activity on re-inoculation into a susceptible variety. In accordance with general experience in human medicine the parasitic activity of *Phytophthora infestans* is thus, within certain limits, increased by passage through susceptible varieties and diminished by passage through resistant varieties.

A similar alteration which, however, must have persisted through sexual reproduction was presumed in *Puccinia digraphidis* (reed canary grass

rust), which lives on Liliaceae in the haplophase and on *Phalaris arundinacea* in the dikaryophase. One of its biological strains, the f. sp. *smilacearum-digraphidis* Kleb., is polyphagous and forms its aecidia equally well on *Convallaria* (lily of the valley), *Maianthemum* (May lily), *Paris* (herb Paris), and on three *Polygonatum* spp. (Solomon's seal).

Starting with this polyphagous initial form, and culturing exclusively on *Polygonatum multiflorum* with occasional returns to *Phalaris arundinacea*, Klebahn (e.g. 1912) tried to produce a race which had, to some extent,



FIG. 150. The pathogenicity of *Bacterium Stewartii* during eight successive passages through a resistant maize variety. *a* control; *b* first infection with a weakly pathogenic culture; *c-f* clinical picture after the second, fourth, sixth, and eighth passage. (After Lincoln, 1940.)

become adapted to this host (habituation race) and which could attack it more readily than the other species of host. After fifteen generations the desired result seemed to have been achieved in so far as the fungus always produced abundant aecidia on *Polygonatum multiflorum*, whereas it attacked *Paris* and *Maianthemum* only slightly. During this time fifteen sexual fusions and reduction divisions had taken place, and hence it was presumed that a gradual adaptation had occurred which was transmitted through the genome; in the neo-Lamarckian sense, an inheritance of acquired characters. But these experiments are not conclusive, because the initial material formed a population, and therefore the objection remains that no change or readaptation of the parasite had occurred but only a selective favouring of those individuals especially suited to *Polygonatum multiflorum*.

(*b*) Increase of virulence in a pathogen by passage through resistant hosts. A weakly pathogenic single cell culture of *Bacterium Stewartii* (p. 169), the cause of maize wilt in North America, produces only a slight disease (Fig. 150 *b*) when first transferred to a resistant variety, but with each subsequent passage up to the eighth its pathogenicity increases (Fig.

150 *f*). If, on the other hand, the same single cell culture be inoculated into a susceptible variety of maize its pathogenicity decreases with successive passages until it reaches a definitive minimum value. The aggressiveness and pathogenicity of *Bacterium Stewartii*, in contrast to *Phytophthora infestans*, are thus reduced by passage through a susceptible host and increased by passage through a resistant host.

(*c*) Alteration of the host range by passage. Passage through uncongenial hosts reduces the vitality of many pathogens and renders them unable to attack subsidiary hosts which are at the periphery of their spectra; to a certain extent, therefore, they become more dependent upon their principal host. In this case, the host range is altered in the negative direction: a partial loss of hosts, contraction of host range, loss of adaptation to former hosts.

In experimental attempts to alter the range in a positive direction (host gain—can a pathogen become habituated to a new host by the appropriate passages?) the results vary with each species of pathogen.

Positive results were obtained, for example, by transference of *Phytophthora infestans* from potato to tomato. Direct infections by *P. infestans* from potato leaves on tomato leaves were occasionally very successful although sometimes only slightly so (Röder, 1935). Yet it is well known that tomatoes planted on the lee side of potato fields infected with *Phytophthora* become gradually and ever more severely attacked by blight. Has the fungus become progressively adapted to tomatoes or have the tomatoes selected those fungal individuals which are able to attack tomatoes equally as well as potatoes?

TABLE XXXIV

The habituation of Phytophthora infestans from potato on the tomato.
(After Mills, 1940.)

Culture	Sporangial formation		Clinical picture 15 days after inoculation
	Old leaves	Young leaves	
Original .	1	0	Few dry lesions, 8–10 mm. long.
Second passage .	2	1	As above; still no stem infection.
Fourth passage .	3	2	Severe leaf infection; stem infection not yet lethal.
Seventh passage .	4	3	Plant dying.

0 = no sporangial formation. 1 = few isolated, 2 = numerous, 3 = abundant, 4 = very abundant sporangiohores so that the leaf surface appears white.

Mills (1940) transferred two single-zoospore cultures of *P. infestans* from potato to tomato plants. After 66–72 hours at an air temperature of 21° C. there appeared on the tomato leaves small brown streaks which soon changed into glassy, oily flecks. The young terminal leaves were very resistant and bore no sporangiohores, whereas the older, lower leaves bore a meagre development of sporangia (Table XXXIV). If these

zoospores were further transferred on tomatoes the virulence of the fungus strain for tomato increased up to the seventh passage (from the eighth to the twenty-second passage no further increase could be observed), whereas its virulence for potatoes underwent no change. Hence *Solanum Lycopersicum* became a new and fully satisfactory host for the parasite without alienating it from the former host *S. tuberosum*.

Thus the problem of how the tomato *Phytophthora* overwinters is solved; it starts anew each year by the habituation of the potato *Phytophthora*.

Negative results were obtained in the attempt to alter the host range of obligate parasites. Stakman *et al.* (1918) cultured the black rust of rye (*Puccinia graminis secalis*) for 3 years on barley, a subsidiary host, without observing any habituation to this host. Also they tried the black rust of wheat (*P.g. tritici*) for 32 months on rye, again a subsidiary host, with the same negative results. The development vigour of rust fungi can certainly be impeded or weakened by passage through non-congenial hosts (see Table XXXV) but cannot be increased by habituation to these hosts.

TABLE XXXV

The influence of the physiological state of the host on the spore dimensions of rust fungi. Uredospores of black rust of wheat (Puccinia graminis tritici), biotype I, on normal and etiolated wheat plants. (After Levine, 1928)

Physiological state	Length	Breadth
	(μ)	(μ)
Normal plants .	34.8 ± 0.22	20.2 ± 0.08
Etiolated plants .	29.9 ± 0.25	19.9 ± 0.11

Hammarlund (1925) inoculated young leaves of wood barley grass (*Elymus europaeus*) with a clone of wheat mildew (*Erysiphe graminis tritici*) to test whether it would gradually become habituated to this subsidiary host; in nature the infectivity for this host is only about a quarter of that for wheat (*Triticum vulgare*). However, no habituation took place; on the contrary, the vigour of the clone diminished, at first slowly then more rapidly until, in the thirty-seventh generation, the fungus died and was lost.

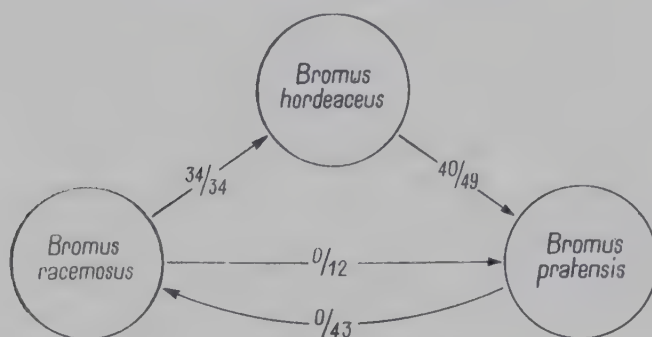
In another series of experiments Hammarlund tried to facilitate the habituation of wheat mildew with the aid of a wound effect. Wheat mildew can attack the leaves of many-rowed barley (*Hordeum vulgare*) through wounds (traumatic infection, p. 54). The question thus arises, can the clone gradually change over to a new diet if it be repeatedly fed through wounds on the cell contents of many-rowed barley? After 128 generations its vitality had not diminished nor had its capacity for infecting healthy leaves increased; as before, the parasite infected fully all wheat leaves but not undamaged barley leaves. A functional adaptation had thus not been induced.

Another attempt was made to facilitate the habituation of the pathogen to a resistant host not by wound colonization but by a so-called foreign

passage, in this case the interpolation of a passage through a slightly susceptible host species. The problem to be solved was whether the pathogen from a highly susceptible host species *A* could be transferred to a resistant host *C*, which it cannot attack directly, if it be first conditioned or habituated to a weakly susceptible host species *B*. The host species *B* is known as a bridging species or bridging host.

In experiments by Salmon (1904), for example, the powdery mildew *Erysiphe graminis* f. sp. *bromi* Marchal from the smooth or bald brome

Infection data from Salmon:



Infection data from Hammarlund:

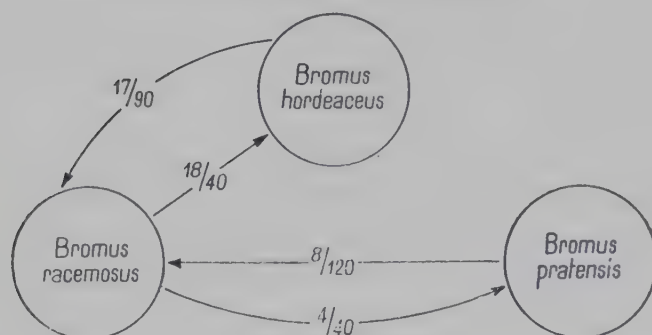


FIG. 151. Diagram of bridging in *Erysiphe graminis*.
Explanation in text. (After Salmon, 1904, and Hammarlund, 1925.)

grass ('traubigen Trespe', *Bromus racemosus*) would not transfer to hairy brome grass ('Wiesentrespe', *B. pratensis*, syn. *B. commutatus*). The numbers in Fig. 151 show how many individuals became diseased out of the total number of experimental plants inoculated. They also show that reciprocal infection did not take place. If he first transferred conidia from *B. racemosus* to soft brome grass ('Gerstentrespe', *B. hordeaceus*) the fungus developed well on these plants and from them, in further experiments, readily attacked the hairy brome grass (*Wiesentrespe*). The soft brome grass (*Gerstentrespe*) therefore constituted a bridging species. Hammarlund (1925), however, obtained different results. Thus, in a few cases with conidial material of high infectivity he succeeded in transferring the fungus directly from *B. racemosus* to *B. pratensis* and conversely. The detour through *B. hordeaceus* means only (as noticed earlier under (a)), that on this host species the fungus attains an especially high vitality whereby,

however, nothing new is produced but there is merely an intensification of what was already present.

(d) The nature of the changes in parasitic adaptation effected by host passage. The alterations in virulence and host range due to the host, so far mentioned, are not only important epidemiologically (p. 168) but have also led to fundamental discussion of the problem of adaptation.

These cases have been interpreted in a neo-Lamarckian sense as adaptations of the parasite to the host and have been cited wrongly as evidence for the inheritance of acquired characters. However, no inheritance of acquired characters is involved because, in the best-established examples, no inheritance occurs. The single cell cultures in question are clones, in the biological sense individuals, which reproduce vegetatively without any new combinations of hereditary material. It is thus a question of individual phenotypic habituation to or alienation from definite functions, e.g. enzyme formation, which do not amount to an alteration of genetic constitution but rather to a heightening of parasitic efficiency through individual training. This increase naturally does not proceed to infinity but only to the limit of parasitic efficiency characteristic of the given pathogen.

On account of their somatic nature these changes in parasitic adaptation are in principle reversible. Nevertheless, in extreme cases, they can become practically irreversible, since once a pathogen has over-passed the downward limit of parasitic efficiency it cannot thereafter effect a return to the host.

Further, true irreversible changes in virulence may also occur in passage experiments. In such cases spontaneous nuclear segregations (saltations, p. 192) or mutations (p. 207) have evidently occurred during the parasitic phase in the interior of the host, i.e. processes of a different order to those envisaged in the adaptation theory.

8. *The Influence of Mixed Infection on the Parasitic Effects of Pathogens*

In some mixed infections the components proceed indifferently side by side; thus rust and mildew on cereals and X- and Y-viruses on potato do not appear to influence one another. In other cases, however, the components may interact as competitors or as synergists, &c., so that the parasitic efficiency of the mixture must be ascertained experimentally in every case.

Two kinds of such mixture are possible: (a) a mixture of different strains of the same pathogen, and (b) a mixture of different species of pathogen.

(a) *The Efficiency of Mixtures of Different Strains of the Same Pathogen*

Can the disease resistance of a new host variety be tested by infection with a mixture of different pathogenic strains of the parasite or must it be tested against the separate individual strains?

In human medicine it is accepted that, in mixed infections, the course of the disease will be determined by the most virulent strain. In infectious diseases of plants, however, this dominance of the strongest pathogenic

strain is not yet fully established, although such cases do undoubtedly occur; for still unknown reasons the relations which obtain are inconsistent.

Three examples, wheat bunt, maize wilt, and barley stripe may be used to illustrate this.

In the case of bunt of wheat (*Tilletia tritici* and *T. laevis*) the efficiency of a mixture of biotypes depends on the current resistance of the host.

TABLE XXXVI

The percentage of bunted wheat plants resulting from infection with single biotypes and with mixtures of biotypes of Tilletia tritici and T. laevis (After Holton and Heald, 1936)

Strain	Hohenheimer wheat	Strain	Ridit wheat
	%		%
1	0.5	1	2.4
5	0	5	2.2
6	0	6	1.4
7a	52.6	7	0.6
13	36.1	92a	50.3
19	0	7a	1
20	0	8	0.6
21	0	9	0.4
23	0	10	2.8
24	0	13	3.2
26	0	16	6.5
28	0	17	2.2
29	0.4	18	3
30	0	19	3
31	0	20	2.4
33	0	21	3.8
34	0	22	3.9
71	0	23	7.2
73	0	24	1.8
78	0	26	9.3
The first 5 strains	13.7 (17.8*)	..	24.5 (11.4*)
The first 10 strains	5.1 (8.9)	..	13.5 (6.5)
The first 15 strains	4.8 (6)	..	11.8 (5.5)
All 20 strains	3.2 (4.5)	..	9.1 (5.4)

* The numbers in brackets are the arithmetic means of the disease incidence for the given strains.

Hohenheimer wheat is very resistant to bunt and, as may be seen in Table XXXVI, is effectively attacked only by biotypes 7a and 13. In the former 0.5 g. of bunt spore material in 100 g. of seed gave a disease incidence of 52.6% and in the latter of 36.1%. If the 0.5 g. of infectious material be made up of equal parts, e.g. of 0.1 g. from 5 different biotypes, the incidence of disease is consistently less than would correspond to the arithmetic mean of the biotypes. The same effect appears if the infection material be composed of 10, 15, or 20 different aggressive biotypes in

equal parts. Thus, in the case of the resistant Hohenheimer wheat, mixed infections of bunt biotypes are less effective than the average of the components.

On the other hand, Redit wheat is considerably more susceptible to bunt than is Hohenheimer wheat (Table XXXVI). When inoculated with a mixture of the same or similar biotypes the incidence of disease is about twice as high as would correspond to the average of the components. The reduced parasitic effect of the mixture of biotypes on Hohenheimer wheat cannot, therefore, be due merely to the mutual competition of the several biotypes but must be based on some as yet unknown antagonism.

The phenomena in North American maize wilt (*Bacterium Stewartii*) (see p. 219) are equally obscure. Diseased plants do not show any characteristic external symptoms, e.g. discoloration, but merely wilt from below upwards, shrivel, and perish. The pathogenicity of different single cell cultures varies greatly (p. 169), ranging from weakly pathogenic strains which evoke scarcely recognizable disease

to strongly pathogenic ones which cause death within 4 days. As a measure of their parasitic efficiency, the following index value is used:

$$100 - \frac{\text{green weight of experimental plants} \times 100}{\text{green weight of control plants}}$$

By green weight is understood the fresh weight of the younger parts of the plant above the first node, 14 days after inoculation. The index covers the range from 0 (avirulent) to 100 (all plants dead and withered after 14 days).

Lincoln (1940) used mixtures, in increasing proportions of a weakly pathogenic strain (pathogenic grade 29) and a strongly pathogenic strain (pathogenic grade about 77), and inoculated them into the first node of the stem of a susceptible maize variety. If the more strongly pathogenic strain determines the progress of disease then each successive mixture ought to be equally pathogenic, i.e. as virulent as the stronger pathogenic strain by itself. But, as is shown in Fig. 152, this is not the case. The abscissae represent the percentage proportion of the two suspensions in the mixture, and the ordinates the average pathogenic index of the mixture. In the lower mixtures (containing up to 20% of the more pathogenic strain) the pathogenicity of the mixture increases more rapidly than corresponds to the proportions of the mixture; above this the pathogenicity of the mixture corresponds to the percentage proportion of the two components. A

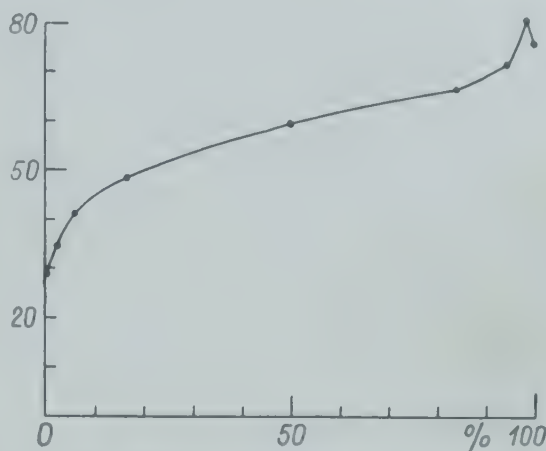


FIG. 152. Pathogenicity of a mixture of two strains of *Bacterium Stewartii* differing in pathogenicity. Explanation in text. (After Lincoln, 1940.)

partial explanation of these facts may be found in the competition of the two strains for living space and available nutrients.

In the third example, leaf stripe of barley (*Helminthosporium gramineum* p. 94), Christensen and Graham (1934) produced 38 fungal mixtures from different combinations of 18 pairs of biotypes and inoculated two varieties of barley with these combinations as well as with the original strains. In most combinations the average disease incidence was equal to that of the strains inoculated separately; the pathogenic effect of their mixture thus corresponded to the average of the components. However, four mixtures diverged from this rule, the incidence of attack being higher than that of the more virulent component.

In none of these biotype mixtures was the parasitic effect below that of the least aggressive strain but over and above this general agreement the significance of the various synergisms and antagonisms still requires explanation. By analogy with experience in human medicine consideration must be given to the possibility that there are strains of pathogen which evoke disproportionately strong defence reactions in the host, thus combining a marked antigenic effect with low pathogenicity. The reduced success of inoculations with mixtures of biotypes, e.g. on Hohenheimer wheat, may perhaps be explained in this way.

(b) *The Efficiency of Mixtures of Different Species of Pathogens*

This discussion will be restricted to true mixed infections, in which the different pathogens enter the host at approximately the same time, and will exclude secondary infections in which a primary pathogen opens the way for a successor. The latter case will be discussed in the following chapter in relation to changes in disease proneness due to pre-disease.

In the true mixed infections the synergisms and antagonisms which were described on page 100 et seq., for the saprophytic phase of the pathogen, also continue during the parasitic phase in the interior of the host. The parasitic effect of a mixed infection may, therefore, be quantitatively greater, less, or equal to that of the components (e.g. mycoses of the lemon).

In other instances the clinical picture and the course of the disease do not constitute a summation of the individual effects but a qualitatively new production which cannot be regarded merely as the sum of its components (e.g. Z-virus of potato).

Lemons, oranges, &c., often show necrotic spots which are caused by various *Penicillia* and imperfect fungi. *Penicillium italicum* (Fig. 153, curve 1) and *P. digitatum* (Fig. 153, curve 2) together exert a summation effect (synergism; Fig. 153, curves 1+2), i.e. the lemons rot faster, especially at temperatures above the optimum, if the infection be due to a mixture of the two pathogens than if the stronger component acts alone.

If *Botrytis cinerea*, which by itself is about as aggressive as *Penicillium italicum*, be added to a mixture of the latter with *P. digitatum* (Fig. 154, curve 2), there is no increase compared with Fig. 153, curves 1+2, but

rather an inhibition or inactivation. However, the further addition of *Aspergillus niger* as a fourth component results in a surprising increase (Fig. 154, curve 3).

In contrast to the first example in which mixed infections cause only an increase or decrease of the same kind of disease effect, in the second example entirely new clinical pictures are produced.

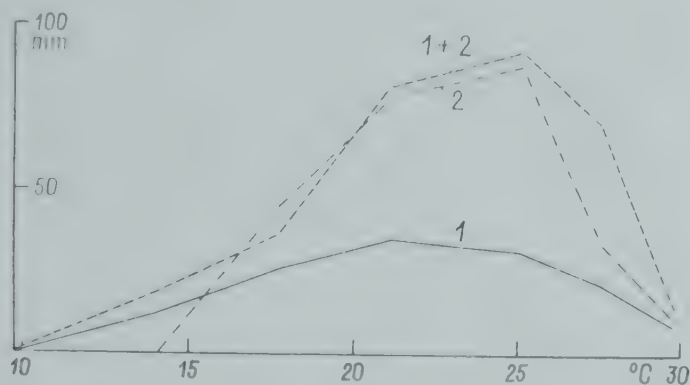


FIG. 153. The influence of temperature on the parasitic effect (diam. of rotting areas after 4 days) of *Penicillium italicum* (curve 1), *P. digitatum* (curve 2) and of a mixed infection of the two fungi (curves 1+2). (After Savastano and Fawcett, 1929.)

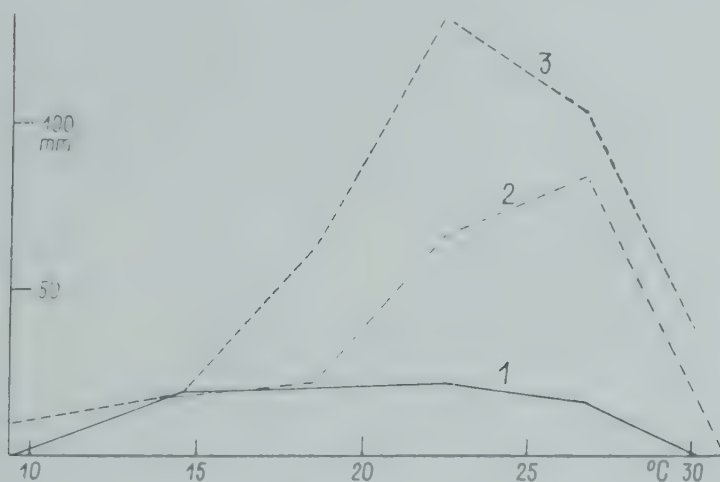


FIG. 154. The influence of temperature on the parasitic effect of some mixed infections (diam. of rotting areas after 4 days). Curve 1: *Botrytis cinerea* alone. Curve 2: *Penicillium italicum* + *P. digitatum* + *Botrytis cinerea*. Curve 3: *P. italicum* + *P. digitatum* + *Botrytis cinerea* + *Aspergillus niger*. (After Savastano and Fawcett, 1929.)

The Z-virus, the symptomless virus of the potato variety King Edward (p. 110), is apathogenic by itself (Table XXXVII), but mixed with the X-virus it produces a 'crinkle', and in mixed infections with the Y-virus, a paracrinkle (Fig. 155); both are extremely severe diseases, which lead to sterility and deformation of affected individuals. These two maladies constitute distinct and new diseases of the potato, which can be reproduced regularly and constantly by the same mixed infections with the given viruses. Only experimental analysis allows us to recognize that the pathogen is definitely not a single entity and, therefore, that the disease is polytopic in origin.

Because of the novel and specific character of the symptoms produced by these mixed infections which, quantitatively, bear no relation to the pathogenicity of the components and which, qualitatively, constitute entirely new productions, it is assumed that the two viruses have produced a complex or formed a chain in which the Z-virus functions as an activator.

However, by taking advantage of their specific paths of infection it is possible to resolve the virus complexes into their components; thus the



FIG. 155. Spontaneous resolution of the ZY virus complex in the potato var. Arran Victory. The stem on the left still exhibits the original paracrinkle (complex ZY), while the stem on the right contains only the symptomless Z-virus. (After Salaman 1932, from Gäumann, 1944.)

Z-virus can only be transmitted to new individuals by grafting, whereas the X-virus is readily transmissible in infected cell sap by needle punctures, thus leaving the Z-virus behind.

TABLE XXXVII

The pathogenicity of three complex-forming potato viruses.
(After Salaman, 1932)

Characteristic	X-virus	Y-virus	Z-virus
Transmissibility . . .	By sap	By sap	By grafting
Symptoms on the potato, var. President . . .	The common mild X-mosaic	The common severe Y-necroses	No symptoms (apathogenic)
Symptoms on <i>Nicotiana tabacum</i> and <i>Datura Stramonium</i> . . .	Symptoms on both differential hosts	Symptoms only on tobacco	No symptoms on either differential host
Pathogenicity . . .	Positive	Positive	Activator only. Intensifies the effect of X and Y

The resolution of virus complexes may also take place spontaneously inside the host plant. The potato tuber shown in Fig. 155 contained the paracrinkle virus complex. It gave rise to two stems, of which the left one still contains the original ZY virus complex and therefore suffers from full paracrinkle, whereas only the symptomless Z-virus migrated into the right branch. In plants which are infected by virus complexes 'new' diseases can appear owing to the resolution of these complexes, i.e. single monotopic diseases corresponding to each virus component; virologists refer to such cases as autoinfections.

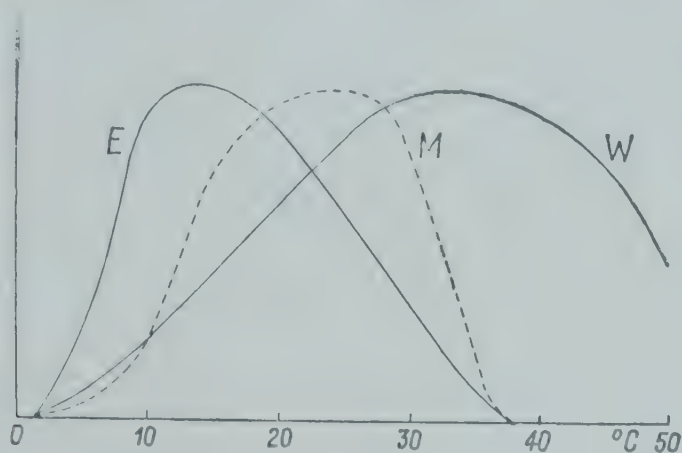


FIG. 156. The influence of environmental temperatures on the growth, enzyme formation, and enzyme action of a parasitic fungus. Curve *M*: dry weight of fungal mycelium at different growth temperatures. Curve *E*: quantity of enzyme per unit dry weight formed at different growth temperatures. Curve *W*: activity of the enzyme at different temperatures. Schematic. (After Gäumann, 1934.)

It is difficult enough in the mixed infections of *Citrus* to apportion responsibility for the different types of effect but the complex-forming viruses provide a total enigma.

9. The Influence of Environmental Temperature on the Parasitic Adaptation of Pathogens

On page 29 et seq., the influence of environmental temperature on the success of infection was considered. The present discussion is a continuation of this and will include the effect on the parasitic efficiency of the pathogen after infection has 'taken' and the pathogen is living inside the host. This problem is specific to botany. When infection is established human pathogens are withdrawn from the influence of external temperature and operate inside the body at a constant temperature as if in an incubator.

It is different for plant pathogens. Because the temperature of plants varies with that of their environment, the temperature at which pathogens inside the host develop their parasitic capacities depends on the prevailing air or soil temperature and on the heat radiation of the sun. These relations are depicted schematically in Fig. 156: firstly, the growth rate of the pathogen depends on the environmental temperature (curve *M*); secondly, the quantity of enzyme formed per unit weight of mycelium also depends on

temperature, but the relation is different from that of growth (curve *E*): fungi tend to produce more enzyme relative to their weight at lower temperatures than at those optimal for growth; thirdly, the enzyme action depends on temperature (curve *W*) but again in an entirely different manner from the curves *M* and *E*: as a rule its optimum lies at a temperature at which the pathogens concerned are generally unable to exist.

If it be now considered that enzyme action also depends on enzyme concentration and that the values of the different functions cannot be summed arithmetically, but are related to one another as power functions, e.g. 1.5^n , and that in each of the more than two dozen enzymes concerned all these curves have different shapes, then the difficulties standing in the

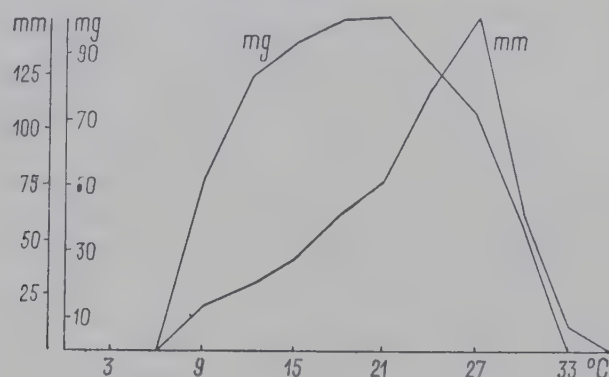


FIG. 157. The influence of temperature on linear growth (curve *mm*) and dry matter yield (curve *mg*) of *Trichoderma lignorum*, strain *A*. Duration of experiment about 20 days.
(After Jaarsveld, 1942.)

way of an elucidation merely of the problem of the temperature effect on the enzyme activities of the pathogen will become a little clearer.

Moreover, the pathogen does not function independently in a vacuum but in continuous interaction with its host. The vital activities of the host are also affected by the prevailing temperature, and usually in a different way to those of the pathogen. Therefore, it is possible to estimate only approximately how much of the parasitic success achieved at a certain temperature is due to the thermally determined efficiency of the pathogen and how much to the thermally induced change in the reactivity and disease proneness of the host. In this discussion only the first part of the question will be considered; the second will be treated in the following chapter.

In the laboratory the temperature requirements of a fungal pathogen are determined from its linear growth-rate or the dry weight of its mycelium. As a rule, the cardinal points found by the two methods are identical, but they may diverge in exceptional cases, e.g. *Trichoderma lignorum* (Fig. 157) which causes a rot of roots, fruits, &c.

For most fungal and bacterial plant pathogens of the temperate zone the minimum temperature is around the freezing-point, the optimum at about 15–20° C. (i.e. approximately the night temperature in summer). The maximum is at about 33–35° C. (i.e. below the body temperature of man; cf. Fig. 158). This upper limiting value corresponds only to the thermal

maximum for vegetative growth, not to the thermal death-point, which varies usually between 44 and 52°C . and depends to some extent on the previous nutrition of the pathogen.

In addition to those pathogens with average temperature requirements, there are others which are favoured by low temperatures, e.g. *Herpotrichia nigra* (Fig. 159) which ceases to grow above 24°C . On the other hand, there are pathogens that are thermophilic, i.e. favoured by warm conditions; e.g. *Gibberella Saubinetii* which causes a seedling disease of wheat, maize, &c.

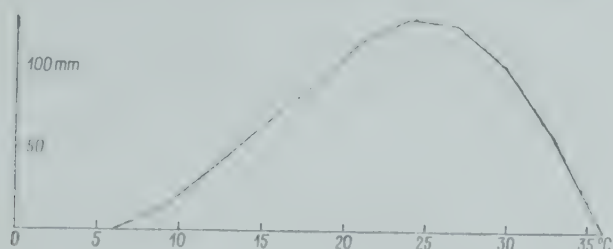


FIG. 158. The influence of temperature on the growth rate of *Corticium vagum* (p. 179). Mean diam. of pure cultures after 117 hours. (After Roth, 1935.)

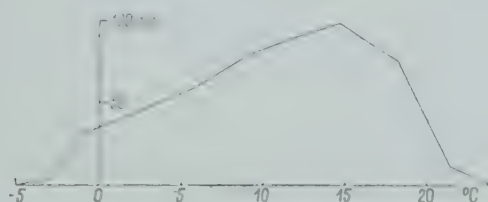


FIG. 159. The influence of temperature on the growth rate of *Herpotrichia nigra* (Fig. 71). Mean diam. of pure cultures after 34 days. (After Gäumann *et al.*, 1934.)

(Fig. 254, curve G), and *Bacterium solanacearum*, which causes a brown bacterial rot of various Solanaceae (tobacco, potato), and also of banana, *Ricinus*, cotton, &c., with an optimum at 35 – 37°C . (Stapp, 1939). Hence, in central Europe, the brown bacterial rot need not be feared, but it is a danger in climates ranging from Mediterranean to tropical.

A difference of about 35°C . separates the minimum and maximum thermal limits for the growth of most plant pathogens which, therefore, are able to utilize the whole temperature range of their environment. A pathogen with a narrower range (cf. Fig. 159) is termed stenothermic (i.e. a special case of stenozoic behaviour, p. 26); if it has a wider range it is termed eurythermic (cf. Figs. 160 and 163).

All other factors being optimal, the optimum temperature for parasitic efficiency of most plant pathogens lies near that for vegetative growth. Thus, in Fig. 160, the difference between the optima for growth and parasitic efficiency is only 2 – 3°C .: *Schizophyllum* decomposes wood a little faster at sub-optimal and a little more slowly at supra-optimal temperatures than would correspond to the linear growth-rate but, on the whole, the two optima coincide. The minimum and maximum for infectivity (p. 24)

are usually somewhat closer than for vegetative growth. In Fig. 162 these relations are shown for the *Gloeosporium* rot of apples.

Because of the specific range optimal for growth of plant pathogenic micro-organisms ($15\text{--}25^{\circ}\text{C}.$) and of the coincidence of the temperature optima for growth and parasitic efficiency, midsummer in our climate is epidemiologically the most favourable period (mean temperatures at Zürich: June, 15.3° ; July, 18° ; August, 21.1° ; September, $13.9^{\circ}\text{C}.$).

In another series of disease agents, which are really the more interesting group biologically, other developmental requirements are not normally fulfilled when temperatures are in the optimal region. Hence these pathogens are unable to utilize their optimal temperatures and their parasitic activity

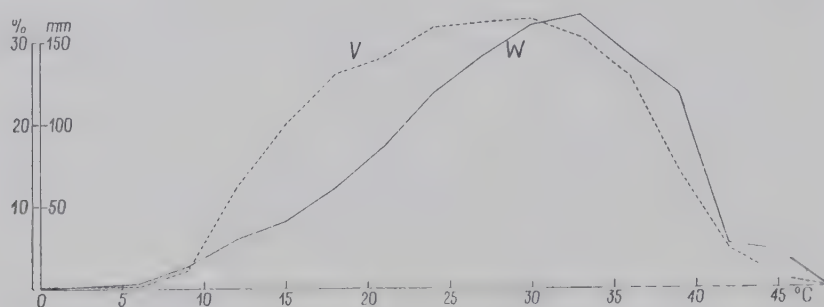


FIG. 160. The influence of temperature on the parasitic efficiency of *Schizophyllum commune*, the cause of a white rot of standing trees (e.g. damaged by sun burn) or of felled timber. Curve *W*: growth rate, diam. of agar cultures after 10 days. Curve *V*: decomposition of sawdust in 105 days. (After Gäumann, 1939.)

is restricted to either higher or lower temperatures. Two examples will be presented for each of the two extremes, i.e. pathogenicity prevailing at very low and at very high temperatures respectively.

The classic example of a parasite that develops its pathogenic activity mainly at low temperatures is *Herpotrichia nigra*, the black snow mould of mountain pine. The rambler in the Alps often finds its black weft covering the branches of the Swiss mountain pine, in avalanche tracks, re-afforested areas, or on shady slopes (Fig. 71).

The development of this fungus depends primarily on the saturation of the atmosphere, and it ceases to grow at a relative humidity below 90%. Development is optimal when saturated air is available for several months at the optimum temperature ($15^{\circ}\text{C}.$, Fig. 159), conditions which are easily produced experimentally. Under these conditions *Herpotrichia* kills the conifers much more rapidly than it does in nature. Hence, any temperature effect on the resistance of the host plays only a subordinate role in the black snow mould disease.

But under natural conditions in the Swiss climate, the fungus never encounters long periods of high atmospheric humidity at optimal temperatures, but only high humidity at lower temperatures under the snow. The temperature-insulating effect (diathermy) of snow is the reason why, in the Alps, the soil under the snow blanket does not freeze (Fig. 161) and why, even 30 cm. above soil level, the temperature in the interior of the snow blanket falls below

—2 to —3 °C. only during particularly cold spells. *Herpotrichia*, however, can still flourish at these temperatures (Fig. 159) and it is, therefore, able to make use of the saturated atmosphere which fills the spaces around the branches of the conifers.

Hence, if conifers in re-afforestation areas are attacked by *Herpotrichia* from the persisting, chronic source of infection, the juniper (*Juniperus communis*), the fungus destroys the young plantations under the snow.

The conditions for *Herpotrichia* attack do not, therefore, include a preference of the pathogen for lower temperatures. The opposite is the

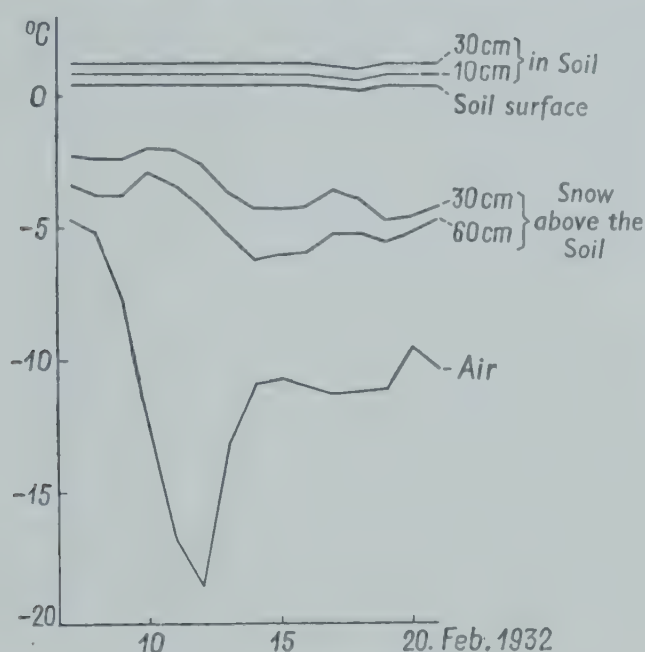


FIG. 161. Course of air, soil, and snow temperatures during a cold spell in Davos; snow cover 65 cm. Slightly diagrammatic. (After measurements by F. Levi and U. Chorus from Gäumann *et al.*, 1934.)

case, but it can endure temperatures round about freezing-point and hence, can utilize the limiting factor of atmospheric moisture where this occurs, i.e. deep enough under or in the snow blanket where the lesions are protected from the severest cold of winter. *Herpotrichia* can, therefore, occur epidemically because of the mildness of the local or micro-climate in the Alps under the snow in winter. In summer, however, its development ceases because the air and sun temperatures are too high and the atmospheric moisture too low.

By contrast, *Herpotrichia* is absent from the foothills, because, as in the mountains, the local climate is too warm and dry in summer and, oddly enough, too harsh in winter. Because of the thin and transient snow blanket a local climate independent of conditions in the outer air cannot be formed around the young plants or lower branches. Climatic conditions, therefore, exert their full effect close to the soil surface. Hence, in winter, neither of the conditions for the presence of the black snow mould is realized in the foothills; the air is too dry and too cold and the

soil and the branches near the ground are frozen to below -10°C . for weeks at a time.

The relative mildness of the winter climate in the high Alps obtains, of course, only under the snow; above the snow blanket circumstances are reversed, as will be shown later in the example of larch canker.

In the second example also, an infectious disease of fruit in cold storage, the pathogen, *Gloeosporium album*, develops from necessity only under the abnormal conditions of the cold store, because here alone its other developmental requirements are satisfied. It is just possible for it to make use of them since the left arm of its temperature curve, like that of *Herpotrichia nigra*, reaches a sufficiently low value.

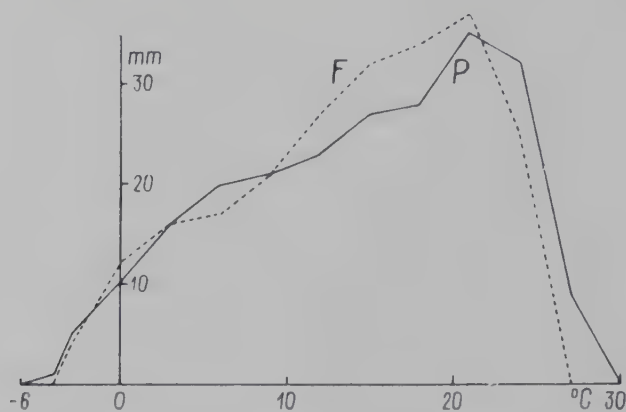


FIG. 162. Temperature requirements of *Gloeosporium album*. Curve *P*: mean diam. of agar cultures after 30 days. Curve *F*: mean diam. of rotting areas on Oetwiler Reinetten apples after 28 days. (Original. Mainly after M. Gruber.)

Gloeosporium album is probably the most dangerous cold storage fungus to certain apple and pear varieties and, with *Fusarium herbarum* (= *F. putrefasciens*) is one of the most important causes of rotting of stored fruit. It infects apples whilst still on the tree and produces necrotic spots several millimetres in diameter, usually with sharply defined margins. Its growth optimum is 21°C . (Fig. 162), whereas that of *F. herbarum* (Fig. 254, curve *H*) extends from 15 to 25°C . At these temperatures both disease agents also develop their optimal parasitic efficiency (Fig. 162, curve *F*). The course of the parasitic relationship is, therefore, determined by the thermally conditioned aggressiveness of the pathogen and not by the thermally conditioned reactivity of the host.

However, at most, these optimal temperatures prevail only temporarily in the autumn and during winter storage. The parasitic success of these two pathogens thus depends on their ability to remain active even at very sub-optimal temperatures.

F. herbarum cannot do this; it ceases to grow at about $+2^{\circ}\text{C}$. and can therefore be controlled by cold storage of the fruit.

Gloeosporium album, however, still grows moderately well round about freezing-point at which, as shown in Fig. 162, its destructive intensity is still a full third of that attained at the optimum temperature. Thus, during

the long months of cold storage, it continues unhindered its parasitic activity on infected fruits and may even reproduce here and so continue the infection chain.

Like *Herpotrichia*, *Gloeosporium album* is also denied any long developmental period at optimal temperature, but it can, like *Herpotrichia* and in contrast to *Fusarium herbarum*, make use of the temperatures at the lower limit of its curve. Thus, it becomes a dreaded scourge of the cold store, a local climate which in this case is man made.

In a number of other infectious diseases, also occurring only at low temperatures, the pathogens are similarly able to avail themselves of the temperatures at the extreme lower limit of their temperature curves; examples are the snow mould of cereals, the epidemic die back occurring in Alpine re-afforestations (Fig. 276), larch canker (Fig. 275) and, rarely, *Phytophthora* rot of potatoes in cold storage (Fig. 196). But in these cases the reactivity of the host is also definitely changed by the low environmental temperature and, therefore, they will be discussed in the following chapter in connexion with changes in the disease proneness of the host evoked by temperature.

The other or right limb of the temperature curve enables organisms to become pathogenic at extremely high temperatures; this happens, for instance, when plant pathogenic micro-organisms spread to man or to other warm-blooded animals.

The problem whether the causal agents of plant disease can also attack man and other warm-blooded animals and vice versa has from time to time aroused considerable interest. Thus, when the 'potato murrain' (*Phytophthora infestans*) swept through Europe 100 years ago, the fear arose that whoever ate of the diseased tubers would also fall victim to the 'epidemic'; this magnified the disaster far beyond the actual reduction in yield (p. 138). The same problem occupied Friedemann *et al.* (1915), who thought they had proved that *Bacterium tumefaciens*, which causes crown gall, was also responsible for infectious intestinal diseases in man; and Friedemann and Magnus (1915), who believed they had induced infectious tumours on potatoes, fuchsias, pelargoniums, &c., using a bacterial strain isolated from a human patient.

A priori, however, an attack by the same pathogen on both human beings and plants must be regarded as an extremely rare event because, as a rule, man is thermally protected from plant parasites, whilst plants are chemically protected from human disease agents.

The body temperature of man is about 37°C., but the temperature maximum for the growth of most plant parasites does not exceed 33-35°C. (p. 230). Chemically, our bodies would be suitable for them, as is shown by their successful growth on nutrient broth, but from the start their development would be inhibited by our high blood temperature quite apart from our defensive reactions. The possibility of such a thermal protection was recognized by Pasteur who asked himself why hens in anthrax-contaminated farmyards did not sicken. Could it be that their high body temperature

(about 42° C.) protected them? By keeping a number of experimental hens with the lower third of their bodies in water at 25° C. he reduced their body temperature to 37–38° C.; on being injected with anthrax bacteria the disease now killed them. But, if the experimental bird was kept in the water bath only until the first disease symptoms appeared and was then taken out and quickly warmed to 42° C., the disease was arrested and the hen recovered (Descour, 1921). Thus, it is really only their high blood temperature that protects hens against anthrax.

Human beings are protected from most plant parasites in the same way as hens are from anthrax, i.e. thermal protection against disease. The fear of Käthi, the grandmother of Jeremias Gotthelf, that the potato murrain could attack her had thus no botanical foundation.

The reverse case, infection of plants by micro-organisms pathogenic to man, fails as a rule through an enzyme insufficiency of the pathogens concerned. Apart from tolerating an acid reaction of the cell sap, &c., a micro-organism must be able to dissolve cell walls, especially the cellulose moiety, before it can attack and destroy plants. However, the common pathogenic micro-organisms of human and veterinary medicine cannot do this and, therefore, *ab initio* plants are chemically protected by their cellulose walls from these organisms.

But, if a micro-organism is both able to grow at 37° C. and to decompose cellulose, it fulfils an important condition for the successful colonization of both plants and man. Two examples will be given.

The epidemiology of the first example has not yet been fully elucidated, although the disease is well known. The organisms involved are the ray fungi or Actinomycetes which cause actinomycosis or 'caries' of the jaw bone. It is usually assumed that, outside man, they live saprophytically on the culms and glumes of cereals and grasses, thus differing from other *Actinomyces* spp. which are true plant pathogens, e.g. *A. scabies* which causes common scab of potato. When such straws are chewed, Actinomycetes pathogenic to man get into the jaw bones, perhaps through carious teeth, where their parasitic development begins at blood temperature. Hence, the disease is twice as common among men as women and generally higher among country folk than town-dwellers.

This infection chain has been disputed and the establishment of proof presents a very real problem; in medicine one is completely dependent in such cases on circumstantial evidence and cannot, as in botany, perform the crucial experiment of infecting serially a few thousand individuals. The highly virulent *Actinomyces* strains are obligate anaerobes and, therefore, are hardly to be expected on grasses. The aerobic or facultative anaerobic strains of *Actinomyces* which have actually been found on grasses are, however, only slightly pathogenic to man, and evoke merely atypical disease symptoms. However, their existence proves that there are strains of *Actinomyces* living on plants whose upper temperature limit is higher than the blood temperature of man; our body temperature does not completely safeguard us from them and we may become slightly diseased.

Thus, it is a controversial problem whether the obligate anaerobic strains of *Actinomyces*, which are strongly pathogenic to man, can also persist temporarily on grasses and cereal ears. The botanist does not regard the obligate anaerobism found in the laboratory as a cogent reason for denying this possibility. Several dozen phytopathogenic bacteria have already been shown to be facultative anaerobes, and it is not known (to the author) whether there may not also be obligate anaerobes living on plants. The colonization of grasses, in the saprophytic phase, and of man, in the parasitic phase, by Actinomycetes which behave as obligate anaerobes

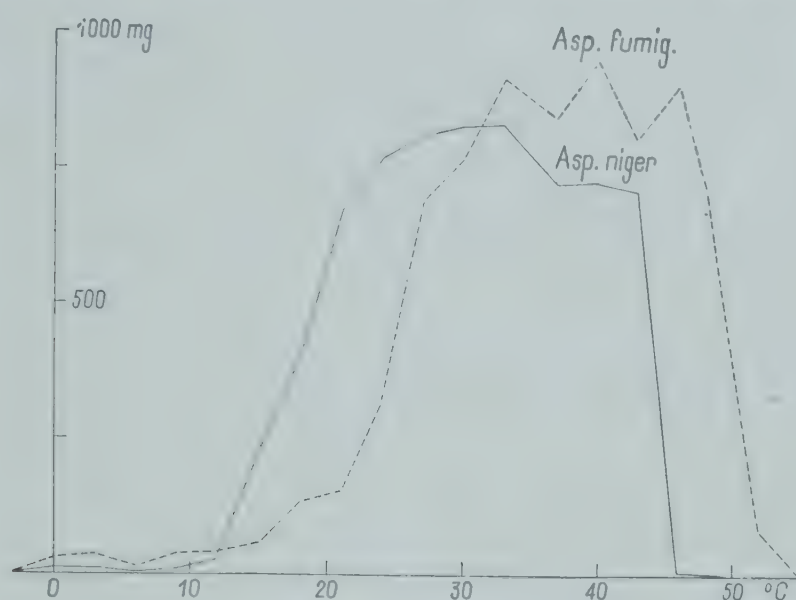


FIG. 163. The influence of growth temperature on the yield (dry weight of mycelium) of *Aspergillus fumigatus* and *A. niger*. Duration of experiment 8 days. (After Gäumann, 1945.)

under laboratory conditions, still remains, therefore, as one possibility among others.

The second is a more clearly cut example; it is the aspergilloses, i.e. infectious diseases of plants, man, and other warm-blooded animals due to Ascomycetes of the *Aspergillus* group. *A. flavus*, *A. fumigatus*, *A. nidulans*, *A. niger*, &c., cause fungal diseases of ears and air passages, e.g. inhalation mycoses of the lungs, &c., in man and other warm-blooded animals. The same species are also plant pathogens causing, for instance, an infectious chlorosis of young maize plants (*A. flavus*, Köhler and Woodworth, 1938), and an infectious disease of maize seedlings present in the dormant seed. If, therefore, farm-labourers sleep in stables where accidentally infected fodder or broken corn, e.g. mouldy maize set aside as cattle food, is lying ready for the next day, the infection chain for 'stable disease' is completed. This also occurs among native populations, e.g. in North Africa, where human beings, poultry, and maize residues are sometimes all housed together in the same room. Such a direct linkage between the aspergillosis of maize and poultry was observed, for example, by Savage and Isa (1933) in Canada, where *A. fumigatus* had

been introduced with maize silage into a poultry farm; it caused pneumonia in 400 chickens with a mortality rate of 90%.

The temperature conditions for a joint attack of plants and warm-blooded animals are seen in Fig. 163. Here the two *Aspergilli* shown are not only extremely eurythermic (temperature range of *A. fumigatus* 58° C.; of *A. niger* 51° C.) but their temperature optima are also very high (between 33° and 46° C. in *A. fumigatus* and between 21° and 43° C. in *A. niger*). Hence, the blood temperatures of both man and poultry are included within their range.

The *Aspergilli* are, therefore, good examples of that rare group of micro-organisms which can attack both plants and warm-blooded animals, the former because they are unprotected chemically by their cell walls, &c., and the latter because they are unprotected by their blood temperature.

§ 3. The Conditions of Parasitic Adaptation of Pathogens

To become active parasites, plant pathogenic organisms must be able (1) to effect entry into the host body and to assimilate the available nutrients, (2) to tolerate or overcome the resistance of the host, and (3) to induce disease in the host by their metabolic products.

1. Entry into the Host Body

Since every single plant cell, in contrast to animal cells, is surrounded by a cellulose wall, a micro-organism must at least be able to dissolve cell walls if it is to attack plants; normally only viruses can pass through plasmodesmata, whose diameter is about 0.1–0.2 μ . This requirement led to a controversy which, historically, is of some interest. Only relatively few bacteria are able to decompose cellulose and to tolerate the acid reaction of the plant cell sap (about pH 5–6) and, for a long time, it was thought that none could attack plants. As early as 1883 Wakker had shown a bacterium, *Pseudomonas hyacinthi*, to be the cause of the yellow disease of hyacinths in Holland; his work, however, was not generally accepted. Even in 1897 Alfred Fischer stated in his *Vorlesungen über Bakterien* (p. 131) that no bacterial plant diseases existed or could exist; that stomata comprised the only connexion of the uninjured plant with the environment and that even should bacterial germs reach the intercellular spaces by this route they would there starve to death. After injury the damaged tissues are soon cut off by a layer of wound cork and the injured parts dry and shrivel. 'Therefore it is not even threatened with bacteria from wound infections and their further distribution in the plant is equally impossible.' This view was opposed by Erwin F. Smith (1899), and for some years a discussion was carried on which finally ended in Smith's favour: there are genuine plant pathogenic bacteria which tolerate the acid reaction of the cell sap, dissolve the cellulose walls, and assimilate the cell contents.

However, the fact that a botanist of Alfred Fischer's standing could go so far as to dispute the existence of bacterial plant diseases on theoretical

grounds, indicates how rigorous is the selection exerted on micro-organisms by the first condition of aggressiveness, the ability to decompose cellulose.

But even a micro-organism possessing this ability cannot, as a rule, penetrate the unchanged cell walls in their natural state by means of the cellulase it produces. Often the cellulose skeleton is impregnated or overlaid with hemicelluloses, pectins, and frequently also lignins, &c. Thus, apart from cellulase, these micro-organisms must produce a whole series of cell wall decomposing enzymes in order to penetrate the host, e.g. protopectinases, ligninases, &c. (Baldacci, 1942, p. 87 et seq.). In this connexion then, plant pathogenic micro-organisms are true specialists as compared with any saprophytic forms and their capacity to penetrate the cell walls of the host fulfils the first requirement of their adaptation as parasites.

However, once the cell walls have been penetrated and decomposition of the more nutritious cell contents, carbohydrates, proteins, &c. has begun, the plant pathogenic micro-organisms need not differ from saprophytic forms either in their special enzymatic faculties or in their nutritional requirements.

Consider first the enzymatic faculties. Plant pathogenic micro-organisms can doubtless manage with the two dozen or so common enzymes (cf. Mayo, 1925; Spitzer and Diehm, 1931) which are also possessed by the saprophytic forms. However, this is difficult to prove, because some enzymatic faculties in organisms may remain latent and be developed only when the pathogen reaches a certain degree of vitality, or when the need for the particular enzyme arises.

For example, if green *Penicillia* are cultivated in a solution of mineral salts with filter paper as the source of carbon, they are unable to produce enough cellulase and die of starvation; if cultivated in a pure sugar solution they produce equally small amounts since the stimulus is lacking. However, in a weak sugar solution with filter paper, cellulase production is abundant: here the stimulus is present and the initial supply of readily soluble carbohydrate enables the fungus to acquire sufficient vitality to decompose the cellulose of the filter paper and utilize it for its own nourishment after the sugar has been exhausted. The capacities of micro-organisms are, therefore, more varied than would appear at first glance; nevertheless, the author does not believe that plant pathogens must necessarily possess any special enzymatic faculties in order to be able to decompose cell contents, protoplasm, carbohydrates, proteins, &c.

The same applies to the nutritional requirements of the pathogen, given that the necessary enzymes for the decomposition of the cell contents are present: the problem is, can the pathogen which has penetrated really live on the breakdown products which it obtains by the enzymatic decomposition of the host plant?

These requirements are hardly more specialized in parasitic micro-organisms than in the ordinary saprophytic forms. All perthophytes and facultative biotrophic plant parasites (p. 58) can, therefore, be cultivated in the laboratory on the same standard organic nutrient media such as

malt or oatmeal agar, bean or wheat straw, steamed potato or rice, nutrient broth, &c. On these they all grow equally well regardless of their particular natural hosts and their degree of specialization and their behaviour give no indication of their later parasitic specialization.

It might, of course, be objected that the above-mentioned laboratory media contain all nutrients in excess and that true physiological differentiation only becomes apparent under the more exacting conditions in the host where certain substances doubtless reach a minimum more rapidly than in our over-rich laboratory media. Clearly, the pathogen within the host is no longer able to choose its food, but must be satisfied with what it finds; surely this food is not so finely differentiated as to explain the host range or the biological specialization of the pathogen.

Thus in wheat there are serological differences between the globulins of the *monococcum* (einkorn), *dicoccum* (emmer), and *vulgare* (bread wheat) groups (Edgecombe, 1931), but there is no evidence that the sharply specialized *Helminthosporia* (p. 189) react to these subtle differences; probably their enzymes break down all these globulins regardless of the detailed structure of the protein. Therefore, in the case of refractory wheat varieties, specific proteins and carbohydrates cannot be the lock to which the *Helminthosporia* have no key.

It is likely that our ideas regarding this problem have been too much influenced by the rust fungi which, it has been suggested as a working hypothesis, are obligate biotrophs and extremely highly specialized just because they can live only on specific, ephemeral (unstable) intermediate substances formed by the host during assimilation. The author does not accept this explanation. Instead of rust fungi, which are ill adapted to experimental purposes, it would be better to use the apple scab fungus which is at least as sharply specialized (p. 189) as the cereal rusts but of which all the biotypes grow equally well as saprophytes on dead leaves of all apple varieties. Hence, all the nutrient materials required by all the biotypes are present in the leaves of all apple varieties, regardless of whether or not any given biotype can colonize the leaves in a living condition. To retain the metaphor of the lock and key, each biotype has an enzymatic master-key to all apple varieties.

Nevertheless, since the majority of biotypes cannot colonize the living leaves of most apple varieties, the cause must lie elsewhere. Hence, the biological specialization of plant pathogenic micro-organisms does not depend in the first instance on nutrient requirements: the chemical fitness of host varieties as substrates is far less specialized than the host selectivity of pathogens.

2. *Interaction between Pathogen and Host*

If a micro-organism possesses the enzymatic key which fits the lock of the host, and if its nutritional requirements are met by the host, it is still not qualified to be a parasite in the living host but only to be a saprophyte on the dead tissues.

The ergot fungus (*Claviceps purpurea*) thrives on dead bean stalks, but in spite of possessing adequate chemical faculties, it never succeeds in colonizing living bean stalks. Special conditions must, therefore, prevail within the cells of living bean stalks which oppose colonization by the ergot fungus and one can only imagine these to be of the nature of local (cellular) defence reactions of the host (see Chapter 4). A micro-organism qualifies as a parasite on a given host only if it can tolerate or counter these defence reactions. This faculty alone, provided the enzymatic and nutritional conditions are met, makes it possible for it to become a parasite and enables it to inhabit the living host.

The interaction with the host is undoubtedly one reason why some pathogens find so much difficulty in changing from the saprophytic to the parasitic mode of life (p. 213). Chemically, no substantially different reactions and capacities are required of them, but biologically they become dependent on an alien life, i.e. they pass, as it were, into parasitic captivity. Their parasitic career will be successful to the degree in which they can adapt themselves to the living activities of the host.

No comprehensive account of this interplay is yet possible. If it was difficult to assess accurately the potentialities of any given pathogen in the sphere of enzyme action because latent, dormant capacities may be released during the course of pathogenesis, how much more difficult must it be to reconstruct the interaction of two living organisms in a planned laboratory experiment.

The finer differentiation of host selectivity and of biological specialization is also to be understood only in terms of this interplay within the living, acting and reacting host.

For instance, it would be almost impossible to describe the preformed (existing prior to infection) specific structural differences of the many thousands of wheat races with sufficient precision to explain the biological specialization of the equally numerous races of rust, smut, *Helminthosporium*, mildew, &c., which exist and continuously throw up new forms intersecting chaotically in their host spectra. It is all the more difficult as the infecting types and the host ranges vary with external conditions. Nevertheless, the biological races of the pathogens first arise in functional interaction with the host; thus, all the races of apple scab can thrive together on dead apple leaves although they are sharply differentiated from one another on living leaves.

The functional defences of the host come into operation initially against the germ tubes. These often penetrate into non-congenial plants and then come to a standstill (Fig. 135). It might be thought they should die of starvation here, but in many cases they grow perfectly well on these tissues provided the latter are dead; thus they are able to decompose them and to live on the resulting products. The reason for their failure as parasites is to be found in the functional defences of the living plant. The pathogen is preceded by a zone of activity (p. 11) within which defence reactions are evoked; the protective substances diffuse from the protoplasts through the

cell wall skeleton and 'protectively impregnate' it so that, in the case shown in Fig. 135, the pathogen is arrested at the cell surfaces of the sub-stomatal cavity.

The defence reactions of the host must be even more effective if the pathogen comes into direct contact with its protoplasm inside the cells. Only here, in the free interplay of forces, is the full subtlety of the functional differentiation of pathogen and host developed. Neither the pathogen alone nor the host alone represents the die which stamps the form of the biological specialization of plant disease agents, but this form is constantly moulded anew in the interaction between the two partners. And yet, to a large extent, this interaction develops repeatedly in the same way since the range of the capacities involved is determined genetically in both host and pathogen.

As to the cause of the biological specialization of the pathogen it may be asked: Which specializes which, the pathogen the host or the host the pathogen? The answer is that they specialize one another in the course of their interaction.

3. *The Material Basis of the Pathogenic Effects of Disease Agents*

The colonization of the host by the pathogen with its continuous withdrawal of nutrients is necessarily associated with certain disturbing and destructive influences which find expression in the course and symptoms of the disease (Chapter 5). So long as they are restricted to a minimum they are of no further interest here, since they are only direct consequences of the capacity of the pathogen to penetrate the host body and to utilize the available substances for its own nutrition.

In some pathogens, however, the disease effect (p. 176) far exceeds this minimum owing to the formation and excretion of special pathogenic (poisonous, toxic) substances. In perthophytes (p. 60) this capacity is the immediate condition for their aggressiveness; their toxins are produced 'purposively'. In biotrophic pathogens (p. 58) any excessive pathogenic effect is doubtless a 'non-purposeful' and largely undesirable secondary effect of colonization; it is useless to the pathogen and damages the host to an extent incommensurate with its provision of shelter and food.

In mild cases, as in all obligate and some facultatively biotrophic pathogens, the toxic effect depends predominantly on the sensitiveness (sensitivity) of the host. Hence it is specific for given pathogen-host combinations. Thus the metabolic products of this kind produced by some rust biotypes have a stimulatory effect on susceptible cereal varieties but are definitely toxic to hypersensitive varieties (cf. Fig. 199). The active substances are obviously produced in minimal homoeopathic doses and are so intimately involved in the interplay between pathogen and host that their chemical nature is likely to remain obscure for some time.

Another group of facultative biotrophic pathogens, the so-called toxigenic pathogens, lend themselves more readily to chemical investigation. In their metabolism they give rise to extremely potent and active toxins

which, in nutrient solutions, are usually produced only after exhaustion of the sugar: these substances when isolated from the pathogen can evoke the disease symptoms in laboratory tests. It is probable that these toxins are, to some extent, specific in their origin since it is likely that each group of pathogens produces its own special type of toxin, e.g. penicillin, fumigatin, &c. In contrast, their effects are non-specific since different types of substances may give rise to clinical symptoms. The same toxin may also cause typical disease symptoms in plant species which the pathogen is unable to attack, i.e. in plants other than the normal hosts of the fungus producing the substances.

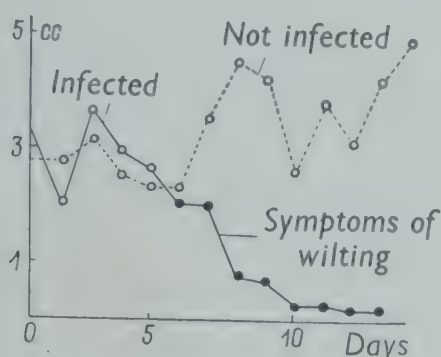


FIG. 164. Mean daily transpiration of healthy cucumber plants (*Cucumis sativus*) and cucumber plants infected with *Fusarium tracheiphilum* (bean wilt disease). Abscissae: days after infection. Ordinates: daily water loss per plant. (After Harris, 1940.)

A beginning has been made in the study of the chemical nature of these toxins, chiefly in relation to wilt diseases, i.e. infectious diseases whose external symptoms resemble spontaneous wilting. They comprise two groups of differing pathogenicity.

One group is represented by brown bacterial rot (p. 63) and maize wilt (p. 169). Here the effect depends on a direct bacterial infection of the host, a flooding as it were of its whole body by the pathogen, rather like anthrax in man. These pathogens are not really toxin forming and, therefore, will not be considered here.

The other group of wilt diseases which interests us in this connexion is caused, for example, by fungi of the genera *Fusarium*, *Verticillium*, *Valsa*, and *Armillaria*. Here the effect is due to an intoxication of the host organism from a local infection (p. 68), such as occurs in tetanus and diphtheria in man. The substances responsible for the disease effect reach the vascular strands from the infection focus and move selectively to particular localities such as the leaves, where pathogenic effects are produced on turgor and other physical properties of the organs, thereby causing the 'wilt disease'.

The wilt effect is very complex and at present is not satisfactorily defined, either histologically or physiologically. For instance, in the tomato wilt disease caused by *Fusarium lycopersici* two extreme types of wilt symptoms can be distinguished: (1) the stem remains upright and becomes somewhat dry although retaining approximately its original diameter; the

leaves show black spots, soon become brittle, and tend to curl at their margins; and (2) the stem bends over, softens, and becomes flaccid, then desiccates, and finally shrivels to a thin wisp; the leaves become much rolled, turn blackish-green to black, and after some time become dry and brittle (Clauson-Kaas *et al.*, 1944).

Physiologically, wilt disease is characterized by a rapid decrease in transpiration (Fig. 164), but similar curves are also shown, for instance,

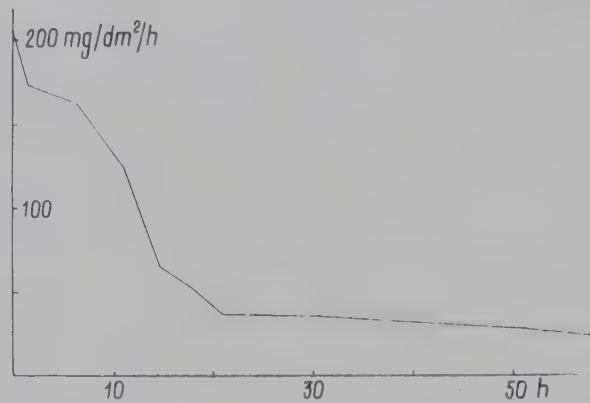


FIG. 165. Effect of a 1% solution of nicotinic acid on the transpiration of a young bean plant at 23° C. air temperature, 40–41% relative humidity, and a light intensity (electric lamps) of 16.2 cal./cm.²/h., corresponding to approximately one-quarter of the maximal intensity of the light of the sun reaching us. Abscissae: hours after the beginning of the experiment. Ordinates: water loss in mg. per sq. dm. stem and leaf surface (reckoned together) per hour. Original.

by rooted bean plants which have been put into a solution of a completely non-specific poison (Fig. 165).

Since 'wilt' is indeterminate and of slight specificity it can be produced by many kinds of substances, e.g. amines, basic amino acids (Lüdtke and Ahmet, 1933), nicotinic acid, and also by culture filtrates of numerous parasitic and saprophytic fungi. The true tomato wilt described above is, however, due to an acidic polypeptide-like compound with the overall formula $(C_9H_{15}O_7N_3)_n$ (Plattner and Clauson-Kaas, 1944) which becomes effective only in the presence of traces of iron. The years immediately ahead are likely to yield much information on the chemistry of toxins and hence on the material basis of the individual pathogenic effects of various plant disease agents.

CHAPTER 4

THE DISEASE PRONENESS OF THE HOST

IN the previous chapter the conditions necessary for the establishment of the parasitic relationship were discussed from the standpoint of the pathogen; they will now be considered from the standpoint of the host. They usually appear as complementary pairs. Thus, the parasitic attributes of the pathogen correspond—like block and print or lock and key—to similar or opposite qualities of the host; for example, the capacity of the pathogen to respond to the stimulus of the host is correlated with the capacity of the host to emit the stimulus, and so forth.

Just as the pathogen requires a congenial host (p. 174), so an affinity for a congenial pathogen is the primary condition for the qualification of a host to serve as a host. Only if a spore chanced to fall on a compatible plant does its germ tube even attempt to colonize it; only in such a case does the problem of the susceptibility or resistance of the given plant arise since, otherwise, the plant is without significance for the germinating pathogen. One cannot, therefore, describe the beech tree as 'resistant' to wheat rust, since the beech has no more existence for the fungus than has a stone.

If the invasion succeeds and infection 'takes', we describe the individual concerned as susceptible to infection or receptive to the pathogen. If, in addition, the infection results in disease, we describe the individual as susceptible to disease. As a rule, the two forms of susceptibility run parallel and, together, form the content of disease proneness; in latent infections, e.g. ring spot mosaic virus on *Melilotus officinalis*, page 182, the host is susceptible to infection but not to disease, i.e. it merely tolerates the disease agent.

If the attempt at colonization fails whilst, under identical conditions, other individuals are infected, the individual in question is termed insusceptible, non-receptive, resistant, or refractory. The two negative terms 'insusceptible' and 'non-receptive' are synonymous, whereas the term 'resistant' does not convey exactly the same meaning; 'susceptible' connotes primarily a passive state whereas 'resistant' implies readiness for active defence.

Moreover, susceptibility or resistance to a given pathogen are not two alternatives but merely two extremes of plant reaction. At the one extreme, complete susceptibility, the parasite spreads freely through the host as if the latter were a neutral, passive substrate; at the other extreme, complete resistance, it cannot develop at all. Between these two extremes lies a transitional range of greater or lesser susceptibility, within which the growth of the pathogen is sub-optimal.

The following can be used as criteria for these quantitative variations in degree of susceptibility.

1. The clinical picture, the type of infection, reaction, or attack. By the method of Stakman and Levine (1922) the reaction types of the host in many plant diseases can be graded in a scale with definite numerals which are now internationally recognized (Fig. 166). Of course, this scheme of assessment needs special revision and modification for each disease, but the following examples will convey a general idea of the gradation.

Type i: immune or free from attack. Infection does not 'take'. The tissues remain completely free from infection and show no macroscopic trace of reaction or discoloration.

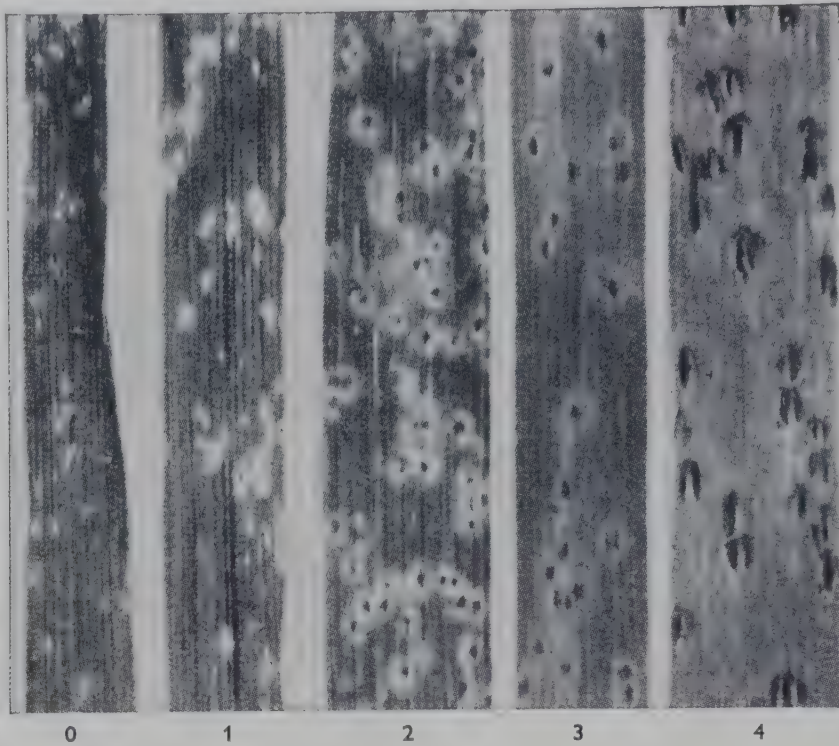


FIG. 166. The reaction types of different wheat varieties to brown rust (*Puccinia triticina*), as an index of their disease proneness. Explanation in the text. $\times 3$. (After Mains and Jackson, 1926.)

Type 0: very resistant. The infection 'takes' and induces in the host a reaction consisting of chlorotic or necrotic spots on which, however, the pathogen does not form reproductive bodies (sub-infections in contrast to full infections; the parasite fails to reproduce itself, so that the plant attacked does not become infectious). This type may be further subdivided into 0o: spots of almost microscopic dimensions (point infections); 0a: spots small, necrotic, brownish, without a central depression; 0b: spots up to 2 mm. diameter with a sunken centre, and so on.

Type 1: resistant. In the centre of the infection area the pathogen forms very sparse fructifications which consist only of imperfect states: minute uredosori in the rusts, small oidial 'cushions' in the powdery mildews, &c. 1a: infection spots of the 0a type; 1b: infection spots of the 0b type, &c.

Type 2: slightly susceptible. The sori of the imperfect states develop more extensively than in type 1, but still do not reach their full size (e.g.

uredosori of some 150–300 μ diameter), and are usually surrounded by a broad, chlorotic halo (reaction zone).

Type 3: susceptible. Moderate to heavy development of sori of imperfect states, these being medium sized, e.g. rust pustules approximately 250–500 μ diameter, with chlorotic-necrotic discolorations of the leaf. At the close of the growing season a few sori of the perfect state (e.g. teleutospores in the rusts) are formed.

Type 4: very susceptible. Extensive outbreak of large sori of imperfect states evenly distributed over the leaf surface, and, at most, surrounded by a weak chlorotic reaction zone. Luxuriant growth of the perfect state at the close of the growing season.

Type x: variable host reaction. Reaction type ill-defined: reaction areas of more than one reaction type are induced on the same leaf surface by a single spore isolate of the pathogen.

This scheme of assessment provides the most delicate criterion for the determination of the host's proneness to disease. It combines both partners, the course of the disease, i.e. the degree of sensitivity of the host, and the penetrative ability of the pathogen, which in its turn is measured by density of sporulation. It can, however, only be applied to highly differentiated pathogen–host pairs and, even here, only to infection by pure biotypes. Simpler criteria are applied in the determination of proneness to disease in less precisely reacting hosts and in biotype mixtures. Such are, for example:

2. The numerical infection threshold: if, *ceteris paribus*, some 5,000 spores per plant are needed to induce 60% bunt infection on Jenkins Club wheat and about 100,000, i.e. twenty times as many, on Marquis (Fig. 40), then the former is more susceptible than the latter.

3. The sporulation density of the pathogen: *Ribes nigrum*, the black currant, in which blister rust produces 2,600,000 spores per sq. cm. of leaf surface (p. 183), is more susceptible than *Ribes lacustre* with only 45,000 spores per sq. cm.

4. The pre-sporulation period of the pathogen: *Echium vulgare* (viper's bugloss) is the most susceptible to the powdery mildew of the Boraginaceae (*Erysiphe horridula* f. sp. *echii-myosotidis*) (Blumer, 1933), because the fungus sporulates profusely on it only 6 days after infection (and not merely a few immature spores!) as compared with 9 days on *Omphalodes linifolia*, 25 days on *Cerinthe* spp., and 4 weeks on forget-me-not (*Myosotis* spp.). A potato variety on which *Phytophthora infestans* (late blight) reproduces in 2–3 days is more susceptible than another on which the pre-sporulation period is 7–11 days, provided environmental conditions are equally favourable for the pathogen (p. 160).

5. The severity of the course of the disease: tomato and tobacco plants, in which infection with *Bacterium solanacearum*, the pathogen of slime disease in warmer climates, causes death in a few days, are more susceptible to this disease than wild Solanaceae and weeds (subsidiary hosts), in which the parasite, under the same conditions, merely evokes a mild, lingering disease.

6. Reduction in yield: a potato variety which suffers a 50% reduction in yield due to *Phytophthora* is more susceptible to late blight than another in the same field which shows a loss of only 20%, and so on.

These data on the proneness of a host to disease often hold good only for particular organs or parts of the plant and not for the particular species or variety. In potatoes, for instance, the susceptibility of the tubers to *Phytophthora* is not always the same as that of the leaves; in var. Flava the foliage is more susceptible than the tubers, in var. Ackersegen the tubers are more susceptible than the leaves, and in var. Bintje both are about equally susceptible. Similar divergences occur among cereals in regard to the infection of their foliage leaves, haulms, and ears by rusts, stripe diseases, &c., so that they do not show a unitary susceptibility (of particular varieties or individuals) to a given parasitic race, but a graduated, specific susceptibility of the different parts or organs. In such cases a choice of evils must sometimes be made in the selection of a variety for cultivation.

Moreover, a certain relativity is inherent in all data concerning the degree of proneness to disease in a host: they are not absolute values but hold good only for the conditions under which they were obtained, since the pathogen (p. 192) as well as the host can modify its disposition under certain environmental conditions.

The more delicate the criterion, the more sensitive it is to external influences; thus, the above-mentioned scheme of assessment for rust biotypes, &c., yields comparable, reproducible values only under strictly identical experimental conditions. For the determination of susceptibility in new varieties (and also of the efficacy of new plant-protectants, &c.) the method of testing should be standardized as rigidly as possible by instructions regarding the environmental conditions of experiment and the strains of the pathogen to be used.

In order, therefore, to simplify the task of applying laboratory results obtained in this way to cultivation on a field scale, one attempts to make the test situation resemble practical conditions as closely as possible. This aim, however, can be realized only to a limited extent since it may happen repeatedly that a test variety sickens under laboratory conditions but not under those of particular areas of cultivation, and vice versa. In varietal susceptibility trials, therefore, laboratory tests cannot replace practical field experiments carried out under the climatic and edaphic conditions of different regions of cultivation.

The number of diseases to which any given host is susceptible is variable. All kinds of plants are afflicted by infectious diseases and so are receptive to one pathogen or another, but the spectrum of the parasites allowed entry by different species is of varying width. Cereals and potatoes are each attacked by some hundreds of miscellaneous pathogenic species, whereas the horse-chestnut (*Aesculus Hippocastanum*), the beech (*Fagus sylvatica*), and mosses are attacked only by very few; hence, the susceptibility spectrum is wide in the former and narrow in the latter.

In the host the relations between disease proneness and systematic affinity resemble those described in reverse for the aggressiveness of pathogens (p. 179). In certain cases no correlation exists, so that the closest relationship (e.g. of cereal varieties) does not imply a like degree of susceptibility. Conversely, in other instances, there exists a strict systematic correlation of susceptibility with certain orders or families, or with certain genera, or with certain species, varieties, races, &c. Thus, in central Europe only ferns, Querciflorae and Rosaceae, are susceptible to the fungus genus *Taphrina*; wheat (*Triticum*) but not rice (*Oryza*) succumbs to rust diseases although both are widely cultivated annual crop plants. Hence, increased discrimination in the host selectivity of the pathogen (p. 184) corresponds with a similar degree of refinement in the susceptibility of the host.

In many plant groups, therefore, disease proneness in its most highly developed manifestation (e.g. apple varieties towards apple scab, p. 187; cereals towards rust, p. 190), is much more sharply specialized than in man. In man a race-linked susceptibility to disease scarcely exists, although, for example, negroes may possibly acquire a slighter and shorter immunity to smallpox than whites. But, in the plant world, the specificity of the hereditary, genotypically defined susceptibility of the host is paralleled, even in its furthest ramifications, by the specificity of the pathogenic biotypes (p. 190).

The present chapter is an inquiry into the nature and material bases of the varying disease proneness of hosts. It falls into two parts.

A. Innate disease proneness; this comprises the inherited, genotypic, constitutional conditions of the susceptible or refractory behaviour of a host.

B. Modifications in disease proneness due to environmental factors, i.e. the disposition of the host including its developmental changes; this constitutes the acquired, modifiable part of proneness to disease.

Strictly speaking, immunity acquired through having suffered from a disease should be ascribed to disposition, since it is not innate but has been acquired during the lifetime of the individual; all that is innate is the capacity to develop an immune reaction. However, the content of immune reactions and of immunity is entirely different from that modification of proneness to disease due to the environment which one ordinarily thinks of as 'disposition'. Immunological resistance to disease is, in fact, acquired in a specific way through the reaction of the organism to the relevant pathogen itself, whereas by disposition is understood only the most general modifications of disease proneness through nutrition, temperature, &c.

A. THE INNATE DISEASE PRONENESS OF THE HOST

Innate proneness to disease is made up of two components:

1. The capacity of the organism to serve as host, i.e. its ability to shelter and nourish the parasite.
2. Its 'inclination' to serve as host, i.e. its ability for functional defence.

Thus, all warm-blooded animals are, in one way or another, capable of serving as hosts to micro-organisms, as is demonstrated by vigorous growth of the latter on bouillon, but they are not so 'inclined' and spontaneously eliminate most infections by their defence reactions.

However, for reasons doubtless rooted in the depths of the human unconscious, we are accustomed to consider the proneness of a host to disease not, in the first instance, from the positive side as capacity and 'inclination' of its organism to serve as a host: 'what is it that renders an organism "sympathetic" to the pathogen?', but primarily from the negative side as incapacity and 'disinclination': 'what means are at the disposal of the organism to render the development of the pathogen difficult or impossible?'

This refractory attitude is seldom due to one factor only, it usually results from the interaction of a whole series of idiosyncrasies and capacities of the given host. The relative importance of these factors and groups of factors differs, although we know little about this. In addition to the main factors which determine resistance, others of subsidiary importance (secondary factors) are involved; if pathogen and host are in a labile state of equilibrium and almost evenly balanced, these secondary factors, which normally play a minor part, may even decide the issue.

Further, the temporal sequence in which these factors operate also varies; an example will make this clear.

TABLE XXXVIII

The relations between the structure of 2-3-day-old leaves of certain Berberis species and their resistance to Puccinia graminis. (After Melander and Craigie, 1927)

<i>Species</i>		<i>Thickness of outer epidermal wall and cuticle</i>
		μ
Highly susceptible		
<i>Berberis canadensis</i>	. .	0.88
<i>Berberis dictyophylla</i>	. .	0.82
<i>Berberis vulgaris</i>	. .	1.10
Slightly susceptible		
<i>Berberis brachypoda</i>	. .	1.43
<i>Berberis Lycium</i>	. .	1.23
<i>Berberis pruinosa</i>	. .	1.16
<i>Mahonia aquifolium</i>	. .	1.30
Not susceptible		
<i>Berberis Thunbergii</i>	. .	1.57
<i>Odostemon repens</i>	. .	1.75

The basidiospore germ tubes of most rust fungi, including *Puccinia graminis* (black rust; p. 84), penetrate through the cuticle into the interior of the leaf in the same way as *Botrytis* (Fig. 1). However, the tough epidermal outer wall and cuticle, which develop on the leaves of the non-susceptible *Berberis* spp. (Table XXXVIII), even in their youngest

stages, act immediately as a mechanical defence: on account of this structural peculiarity the leaves in question are not adapted to serve as hosts to black rust.

On the other hand, in species with more delicate leaves, the germ tubes are able to penetrate into the interior of the leaf, so that in this case the first barrier gives way before them. The highly susceptible members of this group (e.g. *Berberis vulgaris*) have, behind this first barrier, no further possibility of resistance, which is why they are highly susceptible. In other species, however, e.g. *B. pruinosa* and *B. Lycium*, a further group of resistance factors, which may be plasmatic, comes into operation behind the first barrier, and these obstruct the invading parasite; hence, these species are less often and less intensively attacked than the *B. vulgaris* group. Thus, although they are suited, as far as the peripheral leaf structures are concerned, to serve as hosts, they are (to use an anthropocentric expression) 'disinclined' to do so.

In reality, the first factor-complex (the resistance of the leaf surface to perforation) does not come into play initially, to be followed, when it has broken down, by the second factor-complex (the plasmatic defence of the invaded cells) acting independently. On the contrary, the different factors are mutually complementary and, as a totality, decide the outcome of the infection. Through their co-operation, the initial factor-complex hinders the pathogen, so that it arrives 'tired' and weakened in the cell interior, where the second factor-complex then exterminates it. It is not the particular inhibitory substance by itself nor the particular defence reaction, but the interplay of these factors which is decisive and specific for a given host.

When, therefore, in the following paragraphs we analyse the innate proneness to disease into several factors and consider them apart, this analysis has only a methodic and didactic significance; in life it is their totality that is decisive.

The lack of fitness of an organism to act as host is termed axeny, i.e. inhospitableness (*Ungastlichkeit*, *Unwirtlichkeit*, *Inhospitalität*), incompetence, or resistance. The last term is inapposite, since *resistere* in case of war implies active defence by the inhabitants of a region and not that hindrance of the attack by difficulties of terrain which we have in mind here; the term is, however, now firmly established.

The 'lack of inclination' of an organism to serve as host is judged on the basis of its defence reactions; it is an expression of its 'will to defence'.

The difference between these two aspects may be brought out more clearly by reference to the following points of view:

1. Genesis. Axeny is present before infection, prior to the individual becoming a host. In a sense, therefore, it exists in reserve against any pathogen which at one time or another impacts on the life sphere of a given individual and then recoils from the periphery; but this periphery would have been there if the pathogen had not existed, and it first becomes a factor in the axeny because the pathogen recoils from it. On the other

hand, the defence reactions do not begin to operate until after infection, when the individual has already become a host.

2. Character. Axeny is essentially passive or static, based in part on structural barriers (morphological, anatomical, &c.), and leading, therefore, to a protected life behind a barricade. Hence, strictly speaking, an axenic resistant plant is not 'resistant' but merely 'not susceptible'. Conversely, the defence reactions are inherently active or dynamic, i.e. they are a true functional defence.

3. Heredity. Axeny depends on somatic or developmental peculiarities which are inherited as such, and they are, therefore, unalterable outside a certain range of modification (disposition). Conversely, the defence reactions are not inherited as such; it is only the capacity for defence which is inherited and which is correlated with the species. Thus, axeny or 'inhospitableness', like other specific characters, is genetic in nature, whereas defence is a condition evoked by the parasite.

4. 'Motivation'. Axeny arises from characteristics which exist in the plant for reasons independent of pathogens; only as an incidental or secondary effect do they obstruct or preclude the entry or spread of all parasites which attempt to penetrate them by given routes. Thus, the significance of a tough cuticle lies primarily in its relation to water economy and light absorption, but if a pathogen should 'try' to attack foliage leaves of this kind after the manner of *Botrytis*, it would fail. On the other hand, in the defence reactions, the exclusion of the pathogen is directly 'willed'; it is not, therefore, a secondary effect but is itself the content of the given reactions, which are thus more or less 'motivated'.

5. Range of validity. The factors of axeny are non-specific and do not apply originally to a particular pathogen, whereas the efficiency of the defence reactions is more or less specifically limited to the pathogen which evokes them and, perhaps, also to closely allied species. Axeny, therefore, connotes a generalized inhospitableness of the system, whereas the defence reactions are 'directed' towards its protection from a given disease.

A further question arises as to the way in which axeny and the capacity for defence reaction are transmitted from parents to offspring.

Accordingly, we shall divide the section on the innate disease proneness of the host into three sub-sections: I. The material bases of axeny; II. The defence reactions of the host (p. 278); III. The inheritance of disease proneness (p. 356).

I. THE MATERIAL BASES OF AXENY

Undoubtedly the plant structure is by nature very inhospitable, as is demonstrated by the paucity of its germ content even in the absence of defence reactions which, in any case, can hardly occur in the intercellular spaces.

True, intact plant tissues are not completely sterile (Schanderl, 1944), since various fungi, yeasts, and bacteria may be isolated chiefly from fruits and seeds, but also from the vascular tissues of potato tubers. For

example, Marcus (1942) found *Bacillus vulgatus* in 91% of the vegetable marrows (*Cucurbita Pepo*) examined; this bacillus is well known as a parasite of the flower stigmas and evidently penetrates from these into the fruits where, however, it does not multiply to any extent. We may be concerned here with a border-line case between a mere contamination and a delayed or suppressed disease such as occurs in certain other latent infections of fruits and seeds. Usually, under normal conditions, such infections do not develop, but, because of the general axeny of the host organism, remain slight and abortive ('*Raumparasitismus*').

According to the site and nature of the operation of the axenic factors it is possible to differentiate between resistance to infection (§ 1), resistance to penetration (§ 2; p. 259), and resistance to spread (§ 3; p. 262).

§ 1. Resistance to Infection

Resistance to infection comprises every attribute of the host that reduces the probability of infection, everything that prevents access of the pathogen to the host. The plant may, indeed, lie within the host range and so be susceptible, but it evades or escapes the pathogen, much as a person stays at home during an epidemic so as not to become infected. Thus, a plant resistant to infection may be susceptible but inaccessible to the parasite, or only accessible to a limited extent.

Resistance to infection, therefore, does not reduce the disease proneness but merely the probability of attack, the chances of infection. Hence, in infectious plant diseases it is the time factor, i.e. the rate of growth of the susceptible parts of the host, that is of primary importance. This factor curtails either (1) the susceptible stage of development, i.e. the period of development during which the pathogen can penetrate into the host tissues, or (2) the duration of flowering, i.e. the period during which a susceptible floral organ lies open to infection, or (3) it obviates, by means of an appropriate growth habit, the development of microclimatic conditions favourable to infection.

In English, this temporal and spatial outgrowing is termed 'escape': the plant has once more eluded pursuit. In Greek it would be known as *apodrasis*.

1. The Duration of Growth of Susceptible Tissues as a Factor in Resistance to Infection

On a purely statistical basis, if susceptibility remains constant, long-lived individuals and organs are more exposed to infection than short-lived ones; Table XXXIX illustrates this relationship for the attack on certain tropical ferns by *Phycopeltis expansa* (Trentepohliaceae) and by the disc lichen *Tricharia melanothrix*.

On the other hand, if (1) the pathogen has a supply of spore material available only after a given time, or if (2) the susceptible period of growth of the host is limited in time, or if (3) there is a time limit affecting both

pathogen and host, then the chance of their coming together will be lessened and the host's resistance to infection will be correspondingly increased.

TABLE XXXIX

The influence of age on the colonization of tropical fern fronds by epiphyllous algae and lichens. (After Jaag, 1943.)

Test plant	Age of the fronds in days	Proportion of the leaf surface colonized				
		$\frac{3}{4}-\frac{1}{2}$	$\frac{1}{2}-\frac{1}{4}$	$\frac{1}{4}-\frac{1}{10}$	$\frac{1}{10}-\frac{1}{100}$	$< \frac{1}{100}$
<i>Osmunda javanica</i> .	630	+				
<i>Dryopteris megaphylla</i> .	440		+			
<i>Pteris excelsa</i> .	310			+		
<i>Angiopteris evecta</i> .	300			+		
<i>Dryopteris ferox</i> .	197				+	
<i>Histiopteris incisa</i> .	191				+	
<i>Dryopteris callosa</i> .	53					+
<i>Blechnum orientale</i> .	12					+

In the first case, i.e. where the pathogen has a store of inoculum available only after a given time, practical agriculture seeks, by suitable varietal selection or special cultural methods, to adjust the development of the endangered crops so that, as far as possible, it takes place before the production of spores by the parasite, thereby resulting in the field becoming resistant to infection.

Thus, it is only at a certain age that potato plants become susceptible in an economically significant degree to late blight (*Phytophthora infestans*). This stage (curve *Phyt.* in Fig. 167) is, of course, reached earlier in the season by the early than by the late varieties, and it coincides, moreover, with that period of individual development during which the curve of yield rises most steeply. An epidemiologically effective supply of *Phytophthora* spores first occurs in central Europe at the beginning of July. If, therefore, the growth curve of the early variety in Fig. 167 can be advanced by 2 weeks, only a few waves of infection will pass over the field and the damage will be limited.

The farmer, therefore, resorts to the pre-sprouting of his potatoes. He puts the tubers in glass cases in the light to form sprouts before planting-time and only then sets them out; hence at the beginning of the planting season they enter the ground so far advanced in growth that two decisive weeks are gained. Their susceptibility remains the same but the crop matures earlier and, because of this lead in growth, it outstrips the mid-summer waves of infection.

The practice of deep ploughing-under of the stubble of foot-rotted cereals is based on similar premisses; namely, that before the fungi concerned have once more made their way into the upper soil layers the seed-grain of the summer cereal has germinated in the following spring and at least the first stages of its growth do not have to take place in contaminated soil: it escapes infection.

More frequent than the first is the second case in which the susceptible growth-period of the host has a definite time limit, whereas the pathogen has a continuous supply of infective material. In practical agriculture, therefore, an attempt is made to counteract the danger of infection by shortening the period of emergence, e.g. again by pre-sprouting of potatoes, or by using seed of high germination capacity, or by planting at correct depth.

The soil-inhabiting agent of black scurf of potato, *Corticium vagum* (*Rhizoctonia solani*), causes an apical leaf roll of plants infected at an early

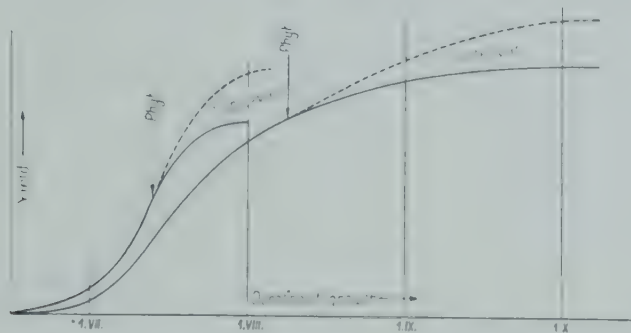


FIG. 167. The yields of an early and of a late maturing potato variety and the time of spontaneous *Phytophthora* infection. Solid curve: actual progress of the crop increase. Dotted curve: expected progress in the absence of late blight. (After K. O. Müller, 1928.)

stage. Sprouts formed in the light are only slightly attacked, so that if pre-sprouted (p. 254) tubers are planted out, the highly susceptible period of growth in contaminated soil is reduced to a minimum.

TABLE XL

The relation between size of grain and attack by *Helminthosporium gramineum* in four-rowed barley. (After Genau, 1928)

Size of seeds (breadth of slot in slotted sieve)	Percentage of infection of a sample from	
	Italy	Dobrudja
(mm.)		
2.5	22.7	27.3
2.2	34.0	35.3
under 2.2	65.9	57.1

In regard to germinative capacity, it is recommended in practice that weak seeds should be eliminated by screening where there is a risk of barley stripe disease (*Helminthosporium gramineum*) (p. 94). In Table XL the smallest grains show the highest incidence of disease. Small grains emerge slowly and irregularly and hence remain exposed to infection for a long time whereas the seedlings of the larger grains, which have a higher germination capacity, outgrow the susceptible stage of development more rapidly. This is also the reason why robust grains from the middle of the barley ear show an incidence of disease only one-half as great as weaker grains from the base and tip of the ear.

Fig. 168 illustrates the effect of depth of sowing on seedling attack by a ubiquitous pathogen; this effect is the same whether the pathogen is applied to the seed before sowing or whether it is introduced into the soil. Seeds that are sown 6 cm. deep take 2 days longer to appear above the soil than those sown 1 and 2 cm. deep, and with increased time and distance the incidence of *Fusarium* is correspondingly multiplied.

Even more common than the second is the third case, in which the availability of both pathogen and host is restricted by certain time limits. The spores of the pathogen may germinate only during a limited period and under given external conditions (before and after, the pathogen does

not 'exist'); in the host the possibility of infection may be limited to particular phases of development or to certain organs, such as unfolding buds, shoots, or leaves (p. 50).

In this case then, the chances of infection are diminished by both partners. If the infection chain is to be carried on in spite of their mutual stenozoia (p. 26), both pathogen and host must make approximately the same ecological demands and possess the same life rhythm, i.e. their growing periods must synchronize and be compatible. For example, any black rust teleutospores (p. 84)

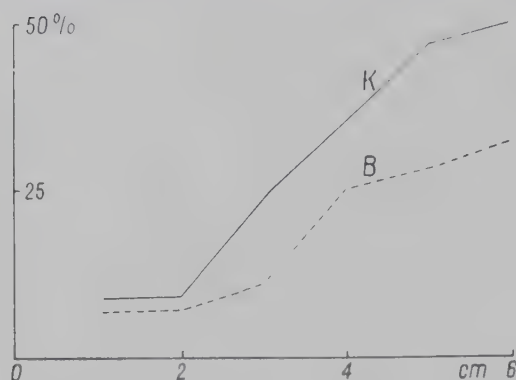


FIG. 168. The influence of sowing depth on the infection of rye by *Fusarium culmorum* (fusariosis). Curve K: seed infection. Curve B: soil infection. Abscissae: sowing depth. Ordinates: disease incidence. (After Baltzer, 1930.)

which germinate before *Berberis* leaves unfold or after they wither merely perish. It is clear that during the Tertiary and Quaternary Periods intensive selection occurred, which only those forms survived that were, in some degree, attuned to the rhythm of the host, i.e. synchronized with it.

This coincidence, however, probably does not go so far as is sometimes assumed, which is why the host plants still exist. The meeting of pathogen and host depends rather on a probability calculus with the aid of large numbers. The black rust teleutospores germinate from February until late summer, producing showers of basidiospores through which the young barberry leaves must grow. The basidiospores germinate, therefore, both much too early and far too late, but under propitious external conditions one of them hits the right moment, induces primary infection, and then begins to spread. It is on account of this low probability of hitting the mark that epidemiologically decisive primary infections are so rarely observable.

In so far as the infectious diseases under discussion are amenable to agricultural methods, these correspond with those of the second group: an attempt is made, through high germinative capacity or correct depth of sowing, to shorten the host's period of exposure and to place it, as far as possible, outside the environmentally determined optimum for the germination of the pathogen. Since, in this, the temperature of the air plays a

decisive part, we shall leave this group of diseases (wheat bunt, &c.) to be discussed in the next chapter in relation to the influence of atmospheric temperature on the resistance of the host to infection.

2. *The Duration of Flowering as a Factor in Resistance to Infection*

The duration of flowering is a determining factor in the probability of infection in those infectious diseases whose route lies through the open flower, e.g. through particular organs (p. 49). This duration involves (1) the blooming time of individual flowers, (2) the rate of fading of the inflorescences, and (3) the flowering period of the field crop as a whole. The shorter the flowering period, the greater is the likelihood that the flowers will escape infection.

This relation should be most obvious in stigma infections, but here the conditions are not as a rule completely covered by a simple correlation of flowering period–frequency of infection, but are much more complicated. Ergot of Gramineae (*Claviceps purpurea*) will serve as an example (p. 75). Its primary host among our cereal species is rye; wheat and barley are subsidiary hosts and, as such, only sporadically and usually less severely attacked. We shall try to account for these differences in infection on the basis of the blooming time of individual flowers, the rate of fading of the inflorescences, and the flowering period of the field crop as a whole.

The blooming time of individual flowers. This is of about the same duration in rye and wheat, 20–35 minutes in the former and 8–35 minutes in the latter, so that the flowers of both species are exposed to the *Claviceps* spores for the same period (only in the case of strictly cleistogamous races of barley, whose flowers remain closed throughout life, is exposure wholly absent and with it the chance of infection). However, both the inner condition of the exposed flowers and the possibility of influencing the blooming period through environmental conditions are different.

In regard first to the inner state of the opening flowers, rye, which needs cross-fertilization, must be effectively pollinated when the glumes spread; hence its stigmas are fully receptive. On the other hand, wheat and barley which are self-pollinated have normally fertilized themselves by the time their flowers open; they have thus entered upon a further stage in their development and it follows that *Claviceps* infection does not 'take' so well in such flowers.

In regard next to the effect of external conditions on the duration of flowering, the blooming times given above for rye and wheat hold good only for ideal cases, i.e. in rye only when fertilization occurs; otherwise, the opening time is greatly prolonged and consequently the glumes remain expanded and the stigmas accessible to infection for days. In wheat, on the other hand, external conditions mostly tend to curtail the period of exposure. The stated blooming time is valid only for fine weather; under cool and damp conditions, in which rye flowers for a long period, the opening of the wheat flower, and with it also infection, are retarded or entirely prevented.

The rate of fading of the inflorescences. If fertilization proceeds normally,

a rye ear completes its flowering in 3–4 days. However, little pollen is available for plants on the windward border of the field, so that fertilization is long deferred or may not occur at all and the ears, therefore, are longer exposed to infection by contaminated flies, &c., than in the middle of the field; further, they are open to spontaneous infections from wild grasses, &c. Consequently, the border plants of rye fields often show particularly heavy ergot infection. In wheat, on the other hand, this artificial extension of the flowering period is absent because the flowers are already fertilized when they open.

The flowering period of the field crop. In rye this normally covers 8–14 days but it is largely conditioned by the density of the stand and the weather. In dense stands, in which the single plants are sparsely tillered, all the ears are in approximately the same stage of development; such a field has a comparatively brief flowering period, so that secondary infections by honeydew conidia (p. 75) come too late. On the other hand, heavy tillering takes place in widely spaced stands and in certain special varieties and continues for a long time in cool, damp weather, resulting in irregular earing and consequently a long duration of flowering over the whole field. Late shoots frequently display heavy ergot infection, since they often remain unfertilized owing to their weakly ears and relatively small amount of pollen, and thus the flowers are exposed for a long time to secondary infections by honeydew conidia from neighbouring ears.

TABLE XLI

The influence of depth of sowing on ergot infection of rye.
(After Wollny from Braun, 1937)

Sowing depth cm.	Rye yield: grain in g.	Ergot yield	
		Number	Weight in g.
2.5	1,391	200	5.3
5.0	1,359	214	8.9
7.5	1,054	575	23.6
10.0	954	947	33.3

Certain agricultural methods work in a similar direction. The greater the depth of sowing (Table XLI) and the width of spacing (Table XLII), the longer, *ceteris paribus*, is the duration of flowering and the higher the incidence of ergot, effects that are all absent in the case of wheat and barley.

Thus, somatic susceptibility to ergot is about equal in rye, wheat, and barley, and the differential incidence of infection depends only on the differential chances of infection due to biological peculiarities of flowering, above all the duration of flowering.

3. *The Growth Habit as a Factor in Resistance to Infection*

The growth habit of a variety or the shape to which a tree is pruned affect the incidence of disease in so far as leafy growth, through overshadowing of the soil, &c., provides a moist local climate (p. 20) favourable

to infection, whereas an open habit facilitates the evaporation of rain and dew, thereby eliminating an important pre-condition of infection. Thus, kidney beans with tall, stiff growth and freely hanging pods (and, of course, still more runner beans) are less subject, *ceteris paribus*, to infection by the

TABLE XLII

*The influence of spacing of the stand on ergot infection of winter rye.
(After Krebs, 1936.)*

<i>Spacing of stand</i>	<i>Number of infected ears per plot of 9 sq. m.</i>	<i>Number of sclerotia per plot of 9 sq. m.</i>	<i>Ergot yield: kg. per ha.</i>
Full quantity of seed (not thinned out)	69.2 ± 1.5	134.5 ± 6.7	5.6
Drill rows 10 cm. apart	115.4 ± 2.2	251.2 ± 3.1	11.2
Drill rows 20 cm. apart	207.6 ± 4.4	494.0 ± 3.8	21.7
Drill rows 30 cm. apart	307.0 ± 8.0	956.2 ± 14.4	34.6

agent of anthracnose (*Glomerella Lindemuthiana* = *Colletotrichum Lindemuthianum* = *Gloeosporium Lindemuthianum*) than squat, drooping varieties; standard roses are less accessible to black spot (*Diplocarpon rosae*) than bush roses; potato varieties with an open habit are less accessible to late blight than compact, close varieties; and so forth.

§ 2. Resistance to Penetration

Resistance to penetration or infection embraces everything in the host that makes penetration by the pathogen difficult or impossible. Hence, resistance to penetration is the equivalent of protection against infection. A plant resistant to penetration may be susceptible but is not accessible.

This distinction is given verbal expression, for example, in Italian. A plant with a tough cuticle is not directly attacked by a given pathogen, it is resistant to penetration and possesses no *recettività* for it. At the same time the inner tissues of the plant may be susceptible and if the given pathogen makes its way through a wound the plant displays a high degree of *suscettibilità* (Baldacci, 1942).

The factors of resistance to penetration form a barricade which halts the pathogen at the periphery. In this way the glumes of cleistogamous cereal varieties keep the smut spores away from the stigmas; infections 'take' only if the glumes are artificially removed.

In general, resistance to penetration, like resistance to infection, operates non-specifically; it is directed indiscriminately against all pathogens that might attempt to penetrate directly from the body surface into the given plant. Three such factor-groups will be discussed here: (1) the structure of the epidermis, (2) the structure of the stomata, and (3) the arrangement of special protective layers with antibiotic substances in their cell content.

1. The Structure of the Epidermis as a Factor in Resistance to Penetration

The plant body is passively defended against a large number of micro-organisms by its superficial covering layers just as the human body is protected by its skin. These layers insulate the plant in both inward and outward directions; transpiration is reduced and disease agents are excluded. This surface resistance to penetration has no significance in those cases in which (with little inner resistance to spread) its protection no longer exists, e.g. in wound infections (p. 54).

Three distinct superficial structures of the plant body may help to impede the entrance of pathogens and so become factors in resistance to penetration; namely, the wax layer, the cutin layer (cuticle), and the cork layers (periderm).

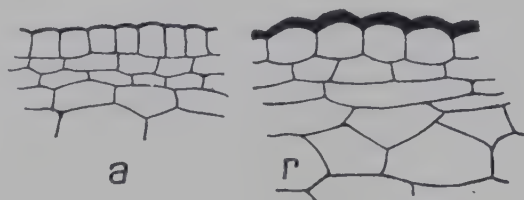


FIG. 169. Section through the peripheral layers of tomato; *a* susceptible to *Botrytis cinerea* (fruit spot disease), and *r* resistant to penetration. The cuticle is black. $\times 200$. (After Ainsworth *et al.*, 1938.)

The wax layer indirectly enhances resistance to penetration by making more difficult the adherence of the infection drop on young cereal leaves, *Prunus* fruits, &c.; that is why, in laboratory inoculations, the leaves of young cereal plants are usually rubbed between the fingers or the surface tension of the inoculum is reduced by chemical methods.

The cuticle provides a direct mechanical resistance to penetration by those pathogens attempting to invade the host tissues after the manner of *Botrytis* (Fig. 1), including *B. cinerea* itself (Fig. 169). It is of special importance in fruits since, with advancing maturity, their inner resistance to the spread of the pathogen falls practically to zero. In the direct infection of fruits only those micro-organisms need be considered which drive an infection wedge through the cuticle. This selection excludes all bacteria and also the majority of fungi since, of the latter, only the highly specialized types are able to adhere by the germ tube (as shown in Fig. 1) and perforate the intact cuticle mechanically. On the other hand, if the cutin layer on fruits is torn or ruptured by hail or insect feeding, or in transport, i.e. if the wall of the safe be forced mechanically, the door is open to any of the unspecialized micro-organisms whose spores are always floating about; hence, injured fruits (and other plant organs) are attacked through wounds by all the ubiquitous fungi and bacteria which could not harm them in their intact state.

Like the cuticle, the suberized periderm of the cortex, roots, tubers, &c. constitutes a direct, non-specific factor in resistance to penetration, even among pathogens using the lenticels as points of entry, e.g. in powdery

scab of potato (*Spongospora subterranea*; Table XLIII). On the other hand, there is no correlation between the thickness of the cork layer and the incidence of infection in the susceptibility of potato tubers to *Phytophthora* since this infection takes place primarily through the eyes, wounds, &c. (Löhnis, 1925).

TABLE XLIII

Correlations between the thickness of the periderm and infection of the potato variety 'Kaiserkrone' by Spongospora subterranea (powdery scab) in crumbly field soil with an abundance of humus. (After Wild, 1929.)

Experimental field	Diseased tubers %	Thickness of the periderm in μ	Number of cell layers of the periderm
Witzwil II. . . .	82.8 ± 6.8	53.0 ± 2.3	2.87 ± 0.01
Clavadel	74.4 ± 3.0	52.8 ± 2.4	2.90 ± 0.01
Witzwil I	64.4 ± 3.2	46.8 ± 3.0	2.60 ± 0.01
Uznach	17.8 ± 6.7	47.5 ± 4.1	2.57 ± 0.01
Regensberg	15.8 ± 4.6	42.2 ± 2.1	2.47 ± 0.01
Wallikon	11.2 ± 2.4	74.4 ± 2.0	4.27 ± 0.01
Regensdorf	2.7 ± 0.8	87.2 ± 4.0	4.97 ± 0.02
Oerlikon	1.9 ± 0.5	83.6 ± 5.2	3.97 ± 0.01
Wagenburg	1.7 ± 0.5	98.0 ± 6.3	4.93 ± 0.02
Schirmensee	1.6 ± 0.3	118.5 ± 13.5	5.67 ± 0.02

2. The Structure of the Stomata as a Factor in Resistance to Penetration

It is probably only against bacteria (p. 15) that the structure of the stomata has any importance as a factor in resistance to penetration.



FIG. 170. The structure of the stomata as a factor in resistance to penetration. 1: *Citrus nobilis*. 2: *Citrus grandis*. $\times 570$. (After McLean, 1921.)

If the bacteria are introduced through needle punctures into the leaf tissue, all *Citrus* species and most of the nearly related genera of the Rutaceae become infected by *Pseudomonas citri*, the agent of *Citrus* canker (p. 152). In field practice, however, certain mandarin varieties (*Citrus nobilis*) show high resistance, whereas certain kinds of grapefruit (*Citrus grandis*) are very susceptible; hence, a problem of simple resistance to penetration must be involved.

The resistant mandarin varieties possess broad lips projecting over the stoma (Fig. 170, 1) and a correspondingly narrow slit-shaped passage leading to the cavity itself, so that dew and raindrops with their contained bacteria scarcely ever reach the mouth of the stoma and the air chamber; the bacteria, therefore, are entirely dependent on wounds as portals of entry. On the other hand, in the susceptible grapefruit varieties, the

lips of the outer stomatal cavity are but little developed (Fig. 170, 2) the cavity stands wide open and literally sucks the water drops down into the mouth of the stoma.

3. *Protective Layers with Antibiotic Substances in their Cell Content as a Factor in Resistance to Penetration*

Housewives prefer brown-skinned to white-skinned onions because they are less liable to 'rot' (onion *Rotz*).

Together with the brown pigment (probably quercetin), there are present in the cell sap of the former certain fungicidal phenols, such as catechol and protocatechuic acid (Angell *et al.*, 1930). Any fungi that attempt to penetrate the brown skin in the same way as *Botrytis* encounter the toxic substances in the cell contents and are inhibited. Thus, the germination and growth of *Colletotrichum circinans* and *Fusarium cepae* are inhibited by the foregoing substances even in dilutions of 300 and 1,200 parts per million, respectively. Thus, for chemical reasons, onion varieties with brown skins are resistant to penetration by these rot-inducing pathogens and suffer less from them than do the white-skinned varieties: the pathogens are dependent on accidental cracks and wounds in the skin as points of entry.

On the other hand, should an onion parasite make use of another path of entry, circumventing the brown skin by penetrating through the neck of the onion, like *Botrytis allii*, then the toxin barrier naturally fails. The same applies if a parasite is insensitive to these toxins at the concentrations present in the cell sap, e.g. *Aspergillus niger*, the causal organism of black mould (Walker *et al.*, 1937).

§ 3. Resistance to Spread

In most infectious diseases of agricultural crop plants the susceptible and the resistant varieties are infected with equal frequency under favourable external conditions; thus, the hyphae of wheat bunt (*Tilletia tritici*) penetrate the epidermis of both varietal groups with the same facility and, 6 days after infection, no difference is observable in the behaviour of the pathogen in the coleoptile and the leaf sheaths (Woolman, 1930). There is, therefore, no resistance to penetration in the bunt-resistant varieties. Only about the ninth day after infection, when the hyphae encroach on the young leaves, do they begin to lose ground in the resistant varieties, and after a further 50 days the infection foci in the latter are localized in the first three leaves: the fungus has not kept pace with the development of the host and the ears remain healthy, whereas, in the susceptible varieties, the fungus has long since reached the growing-point.

What are the factors involved which cause the pathogen to fall behind in the resistant varieties? Two factor-groups co-operate here: (1) the factors of static resistance to spread which already exist in full measure prior to infection, before the pathogen enters the life sphere of the host,

and (2) the dynamic defence reactions released after infection. In the present section we shall deal only with resistance to spread.

Resistance to spread comprises the hindrances existing prior to infection which oppose the generalization of the invading pathogen and its spread in the tissues of the host: the plant is susceptible to infection but is not habitable. Resistance to spread thus implies inhospitableness *par excellence*: true, the door is open to the pathogen, but occupation of the room is denied to it simply by the nature of the host and not by any positive action on the part of the host.

Although resistance to spread exists prior to infection, it only takes effect after infection. Its efficiency against any given object is difficult to assess because it works side by side with the cellular defence reactions and must share any success with them.

In its material bases, resistance to spread may derive from histological characteristics or from the resistance which the cell walls oppose to the progress of the pathogen through their mechanical properties or chemical structure. This resistance is (1) distributed throughout the tissues, or (2) localized in certain barricade tissues.

In other cases, resistance to spread is conditioned by chemical peculiarities of the cell contents, e.g. by an insufficiency of nutrients or an excess of noxious substances; therefore (3) nutrient content, (4) growth substance content, (5) acidity, (6) osmotic relations of the cell sap, (7) antibiotic substances in the cell content, and (8) pre-formed antibodies, are factors in resistance to spread.

1. *The General Resistance of the Cell Walls to Spread*

One instance of unligified and one of ligified tissues will be discussed.

The cell walls of the ground tissue of potato tubers are usually mechanically perforated and not chemically dissolved by the hyphae of *Pythium de Baryanum*, the agent of a dry rot, and, hence, the varying resistance to spread of the different potato varieties depends mainly on the resistance of their cell walls to perforation (Hawkins and Harvey, 1919). For example, in two susceptible varieties the hyphae advanced respectively 366 and 436 μ in 24 hours whereas in a resistant variety they advanced only 102 μ ; to make their way through a host cell they took respectively 43 and 50 minutes in the former varieties whereas in the latter they took 204 minutes. Thus, the cell walls of the latter are perforated more slowly. They were further shown experimentally to exert greater resistance to purely mechanical pressure. The perforation of the cell walls of the skin demands pressures of 48.7 and 42 kg. per sq. cm., respectively, in the susceptible varieties and of 89.4 kg. in the resistant variety; in the interior of the tubers (for the ground tissue) 31.3 and 38.2 kg., respectively, in the susceptible varieties and 65.9 kg. in the resistant variety. Similarly, the crude fibre content of the skins amounts to 1.97% in the former compared with 3.27% in the latter, and that of the ground tissues to 1.9% in the former as against 2.15% in the latter. Thus, the variety resistant to spread differs

from the susceptible varieties by a more extensive secondary thickening of the cell walls.

In the lignified tissues, the lignification in the first place constitutes a general resistance to the spread of all those fungi which do not form ligninases, e.g. the blue-staining organisms of standing and processed wood which are, therefore, dependent on the medullary rays, pits, &c. (Fig. 171).

In addition to lignification itself, the degree of swelling of the lignified membranes may, on occasion, be a factor in resistance to spread. This aspect is technically important in connexion with the influence of the felling date on the durability (i.e. the resistance to fungi) of spruce and fir wood, but should also be taken into account in relation to the susceptibility to disease of standing timber.



FIG. 171. The growth of hyphae of *Trichoderma lignorum* in the medullary rays and, through the pits, into the tracheids of *Pinus taeda*. $\times 120$. (After Spradling, 1936.)

The mature heart-wood and the young sap-wood of the spruce (*Picea excelsa*) and the silver fir (*Abies pectinata*) do, in fact, vary in their liability to decay (*vermorschbar*) under constant environmental conditions when the trees are felled in different months of the year. The dry rot fungus (*Merulius lacrymans*), the wall fungus (*Polyporus vaporarius*), the cellar fungus (*Coniophora cerebella*), and an agent of 'mussel crack' (*Muschelbruch*) *Lenzites abietina* which also develops on standing trunks as a sequel to sun scorch clearly cause

more extensive disorganization in green trunks felled between February and July than they do in winter-felled trunks (Fig. 172, curve I).

Contrary to expectation, these variations in resistance to spread of wood 'felled in sap' are not primarily the result of differences in its nutrient content due to the time of the year, but to its seasonal growth substance content and the seasonal extent of swelling of its cell walls (Gäumann, 1932).

1. The above-mentioned fungi, though not yet studied in detail, are growth substance heterotrophic, i.e. dependent on the growth substance content of the substrate. When the tree 'come into sap' in the early spring, the trunk is supplied with growth substances from the buds and cambium; its content of heteroauxin, aneurin, biotin, adermin, &c., is, therefore, subject to seasonal fluctuations. In its turn, the higher growth substance content promotes the development of the fungi concerned and with it their destructive efficiency. On the other hand, when the formation of the annual ring comes to an end at the beginning of August, the stimulation of

fungal growth also declines; thus, this sector of the influence of felling time does not run parallel with the growing period but with the rhythm of annual ring formation.

2. The degree of swelling of the cell walls, in particular of the cellulose reticulum, evidently varies in the different felling months. The variable tendency to rot mostly concerns the cellulose and, therefore, the correlation coefficient between the decomposition of cellulose (but not of lignins) on the one hand, and the tendency to decay on the other hand, is usually between 0.7 and 0.9, i.e. very high.

If samples of cut wood are stored dry for a year in a well-ventilated shed, they undergo no loss of substance during this period, but they dry out and their colloidal structures, especially their cell walls, mature and

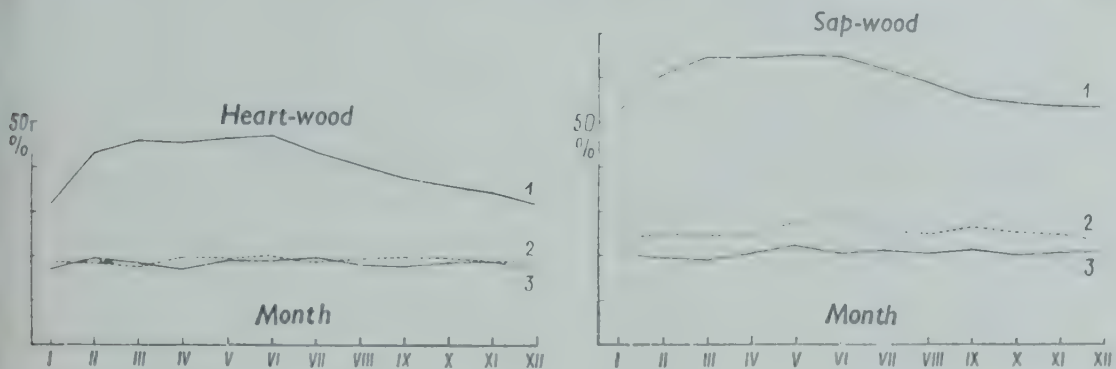


FIG. 172. The influence of felling time on the decay of fir wood by *Lenzites abietina* under constant external conditions. 1 Green wood. 2 Wood stored dry for a year. 3 Wood weathered for a year. Abscissae: felling months. Ordinates: % decay in six months. (After Gäumann, 1930.)

shrink, in part irreversibly (hysteresis). Under the same experimental conditions, the decay of these samples is only half as extensive as in the green state (Fig. 172, curve 2); their resistance to fungi is thus approximately doubled by the hardening of their cell walls. Moreover, the difference between heart-wood and sap-wood is obliterated; the stronger tendency to decay of the green sap-wood cannot, therefore, be determined solely by its higher content of carbohydrates, nitrogen compounds, &c., for it still contains these substances after storage, but the cause of its lower resistance to spread must lie in the great hydration of its cell walls. Finally, the variations in the tendency to decay in relation to time of felling become so slight as probably to fall within the local fluctuations.

In the case of wood samples seasoned in the open for a year, and so exposed to the influence of sun, wind, rain, snow, and frost, there is a still more marked levelling of fungal resistance; not only are they colloid-chemically aged for a year but, over and above this, they have lost about half their carbohydrates and almost two-thirds of their nitrogen compounds through leaching. Their resistance to spread has thereby been still further increased (Fig. 172, curve 3) and the differences between heart-wood and sap-wood and summer and winter felling have disappeared.

Thus, the seasonal fluctuations in the fungal resistance of spruce and

fir wood (but much less of beech wood; Gäumann, 1936) only operate to their full extent when the wood is used immediately in a freshly felled, green condition. If the wood be stored dry for a year, as was the custom of our forefathers, its resistance to spread is appreciably increased by the

colloid-chemical ageing of its cell walls and the differences arising from the time of felling are largely equalized. Further, if the wood be left to weather out of doors for a year (or floated for a considerable time) the seasonal variations are entirely obliterated.

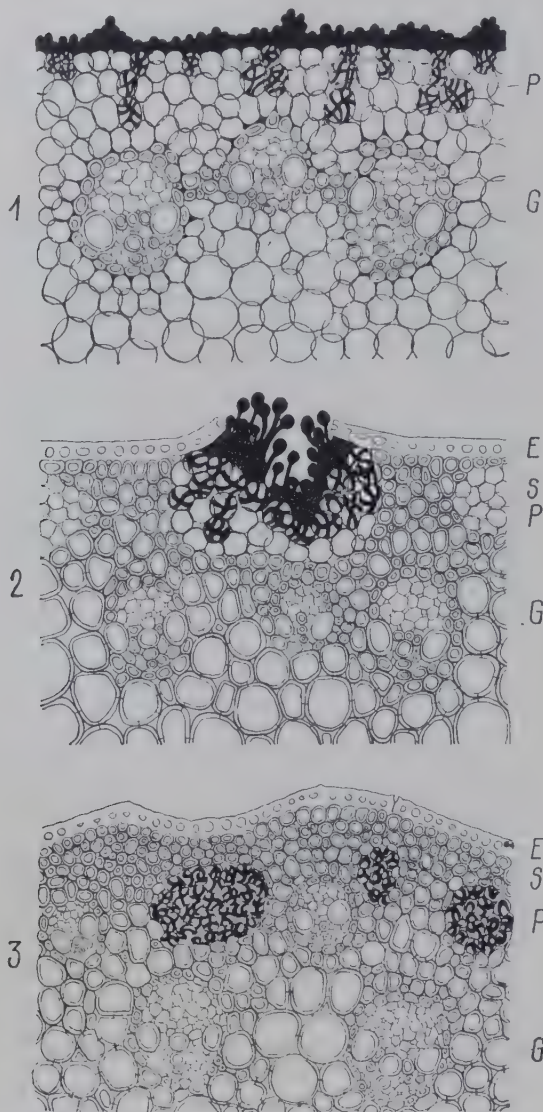


FIG. 173. The resistance of the haulms of three wheat varieties to black rust. 1: *Triticum compactum*, var. Little Club. 2: *T. vulgare*, var. Kota. 3: *T. vulgare* var. Acme. E epidermis. S sclerenchyma. P parenchyma. G vascular bundles. The hyphae and the uredosori in black. Approx. $\times 150$. (Diagrammatic after Hursh, 1924.)

2. Barricade Tissues as a Factor in Resistance to Spread

Whereas the structure and condition of the cell walls discussed in the preceding section create a general resistance to the spread of pathogenic fungi in the tissues concerned, the barricade tissues merely bring about a local resistance to spread within the host organism.

Fungistatic tissues are found particularly among cereal species; thus, the hyphae of the snow moulds (*Fusarium nivale*, *Gibberella Saubinetii*), &c., often spread only through the primary cortex and are arrested by the endodermis, so that they do not reach the key vascular tissues.

In the leaves and haulms the mycelium of the rusts and *Helminthosporium* spp. can spread freely in the chlorophyll-containing tissues (parenchyma and collenchyma) but not in the lignified sclerenchyma, hence the striate arrangement of

many uredosori on the leaves and the name 'stripe disease'.

This histological resistance to spread is particularly obvious in the resistance of wheat haulms to black rust (Hart, 1931). A severe attack on the uppermost section of the haulm which bears the ear causes the maximum reduction of yield. Here the susceptible parenchyma and the resistant sclerenchyma are in inverse proportion. Varieties in which the parenchyma forms a cylinder round the haulm run the greatest risk of black rust and

admit of the most abundant sporulation by the parasite (Fig. 173, 1). Other varieties, in which the sclerenchyma ridges out to the epidermis, dividing the parenchyma into separate columns, allow the parasite only a localized development and a meagre spore production (Fig. 173, 2); consequently their haulms, with an equal plasmatic susceptibility, are definitely more resistant to black rust. Finally, varieties with a still more robust development of sclerenchyma force the susceptible parenchyma inwards and enclose it on all sides by sclerenchyma (Fig. 173, 3), so that only a sparse and usually sterile growth of the pathogen can take place; hence the

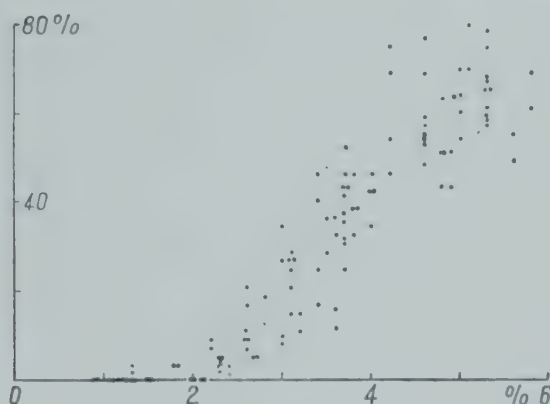


FIG. 174. The influence of the sugar content of pine roots on their susceptibility to mycorrhizal fungi. Abscissae: content of reducible substances (mainly sugars). Ordinates: % short roots spontaneously infected by mycorrhizal fungi. (After Björkman, 1942; from Romell, 1942.)

threatened portions of the haulm in these wheat varieties remain, for all practical purposes, free from black rust.

3. *The Nutrient Content of the Tissues as a Factor in Resistance to Spread*

By its amount and composition, the nutrient content can modify the suitability of the host cells and tissues and so become a factor in their susceptibility or their general resistance to spread.

Tissues rich in nutrient content usually permit a more luxuriant growth of the pathogens than those with a scanty food supply but this relationship obtains only at low subsistence levels. Thus, the susceptibility of pine roots to mycorrhizal fungi (Fig. 184) increases at first with their sugar content (Fig. 174). Hence, all factors such as strong light and deficiency of phosphoric acid and nitrogen (inhibition of protein synthesis) which lead to an excess of soluble carbohydrates, favour the development of mycorrhiza (Björkman, 1942).

However, once the minimum is appreciably exceeded and the available nutrients for the maintenance of the parasite are present in abundance until it has killed the host cells, it is no longer directly benefited by a further rise in the nutrient content; whether fungal hyphae encounter 5 or 15% sugar in the cell sap is immaterial to them (luxury consumption).

Such nutrient provision, however, may well indirectly lower the resis-

tance of the tissues to spread, inasmuch as leads it to a 'fattening' of the cells and thereby to a reduction of their capacity for defence. For example, although the susceptibility of intensively selected fruits and sugar beets to many 'rotting' agents increases with ripening and rising sugar content, it is not the sugar content, as such, that is directly responsible; this constitutes only the cause and the index of the growing incapacity of the cells to react (ballast action).

4. *The Growth Substance Content of the Host Cells as a Factor in Resistance to Spread*

Here we are concerned only with growth substances of the aneurin group, since it is their connexions with the parasitic fungi that are best known, but similar relations no doubt prevail with vitamin B₂ (lactoflavin), with substances of the bios group such as mesoinosit (bios 1), biotin (vitamin H), pantothenic acid, &c., and finally with specific fungal growth substances of the groups M, B, &c. (Fries, 1938; Schopfer, 1939; Janke, 1939).

Some parasitic fungi are completely aneurin-autotrophic and can themselves synthesize the aneurin molecule (vitamin B₁, thiamin), in adequate amount. These fungi include *Ustilago zaeae* (maize smut; Fig. 144), certain graminaceous Ustilagineae, e.g. *U. tritici* (Fig. 98), *Corticium vagum* (p. 179), *Ophiobolus herpotrichus* (foot rot of cereals; Fig. 91), and *Gibberella Saubinetii* (snow mould of wheat). Such fungi are indifferent to the aneurin content of the host tissues. In certain other fungi the capacity for aneurin production is poorly developed; e.g. *Nectria coccinea* (beech and poplar canker), *Valsa ceratophora* (a secondary bark parasite on numerous broad-leaved trees), *Sclerotinia cinerea* (blossom and twig blight and fruit rot of stone-fruit trees), and *Lenzites sepiaria* (a wound parasite on conifers and a general disorganizer of their wood). Consequently, these fungi make only slow growth on aneurin-free synthetic nutrient solutions and are actively stimulated even by a few milli-microgrammes of aneurin.

Still other parasitic fungi are quite incapable of synthesizing the necessary aneurin for themselves. They are partially or wholly aneurin-heterotrophic, so that they must obtain from the host not only their actual foodstuffs but also their specific growth substances and hormones. The vital aneurin cannot be replaced by the general growth substances for the development of flowering plants (auxins and heteroauxin), since these are useless for the growth of most fungi and bacteria. Thus, for a pathogen of this kind, only those plants are susceptible which, from the outset, are able to provide it with the special growth substances from the aneurin group that it needs. Hence, lack of these growth substances is a decisive factor in general resistance to spread.

However, the aneurin requirement of these aneurin-heterotrophic fungi varies in degree. *Phytophthora fagopyri* (root and seedling disease of buckwheat, beans, &c.), *Cercospora herpotrichoides* (eye spot of cereals;

Fig. 92), *Schizophyllum commune* (white rot of beeches and other hardwoods), *Sclerotium Rolfsii* (root-collar parasite of numerous plant species in hot countries), and others are only partially aneurin-heterotrophic. They require only one component, pyrimidin, to be supplied by the substrate, whilst they themselves synthesize the other component, thiazol, and with the addition of pyrimidin can build up the aneurin molecule.

By contrast, *Mucor Ramannianus*, a soil-inhabiting mould, requires only thiazol because it provides the pyrimidin itself.

Slightly more dependent is *Ustilago violacea* (anther smut of Caryophyllaceae; p. 51) which can produce neither of the components itself but can synthesize the aneurin molecule from them if they are placed at its disposal; the test becomes positive at about 0.01 μ g. in 25 c.c. nutrient solution.

The following fungi are absolutely dependent, i.e. wholly aneurin-heterotrophic: *Phytophthora cactorum* (cactus rot, transmissible to numerous other plants), *Phytophthora capsici* (*Capsicum* fruit blight), *Phytophthora cinnamomi* (cinnamon phloem rot), *Trametes radiciperda* (syn. *Polyporus annosus*, dry red rot of standing spruces), *Polyporus adustus* (white rot of beeches), *Ustilago scabiosae* (anther smut of *Knautia* spp.), *Tilletia tritici* (bunt of wheat), &c. These fungi must be provided by their substrate with the aneurin molecule ready for use.

These instances of partial and total aneurin-heterotrophy refer only to the simplest case, namely, a phytopathogenic micro-organism able to manage with a single growth substance, in this case, aneurin. On the other hand, a number of plant parasites require several; thus, for *Lophodermium pinastri* (needle fall of pines), *Valsa pini* (die back of pine branches), &c., a synthetic substrate to be assimilable must contain aneurin plus biotin (synergism).

In the case of *Ophiostoma ulmi* (Dutch elm disease; Fig. 85), aneurin enhances the degree of activity of the essential adermin (vitamin B₆; Fries, 1942). Singly, aneurin, biotin, and mesoinosit each exert only a feeble influence on *Nematospora gossypii* (striate discoloration of cotton bolls), as also does the combination aneurin + biotin, whereas the combination biotin + inosit and still more the combination of all three growth substances increases mycelial development twenty-fivefold. It is to be presumed, therefore, that a highly susceptible host contains all three growth substances.

Moreover, in all problems concerning growth substances the parasitologist is left with the doubt whether the results of laboratory cultures can be applied directly to conditions within the host tissue and, more especially, to the problem of susceptibility. For instance, the capacity to synthesize aneurin can be influenced by nutrition; thus, *Rhodotorula Sarniei*, a wild yeast, is unable to synthesize pyrimidin using glucose as a source of carbon, but can do so if a small amount of glycerine be added. *Pythium Butleri* (root and stem parasite of *Zingiber officinale*) is aneurin-autotrophic on a substrate with a normal salt content (1.64 g. per litre) and asparagin and

glucose; on the other hand, a tenfold increase in the salt content impairs the capacity to synthesize pyrimidin, so that this substance must be supplied (Robbins and Kavanagh, 1938).

In order, therefore, to assess the significance of these varying degrees of growth substance heterotrophy in relation to the problem of general and specific susceptibility, it must be proved by a series of examples that the given host or organ, in both a healthy and a diseased condition, really provides the vital growth substance, whereas the non-susceptible related species or varieties are unable to do so. Since research on growth substances is still in its infancy, this field has not yet been explored.

In this connexion it is natural to reflect on the problem of the obligate biotrophic parasites (p. 177) and to wonder whether they depend on a particularly delicate interplay of growth substances which ceases with the death of the host cells. We do not know.

5. *The Acidity of the Cell Sap as a Factor in Resistance to Spread*

By analogy with the gastric juice in man which, by means of its acid composition affords protection against tubercle and cholera bacilli, it was surmised that the acidity of the cell sap in plants might be a factor in general resistance to spread.

Certainly the degree of activity of the parasite enzymes, as of all enzymes, is controlled by the hydrogen-ion concentration of the substrate, so that no doubt in this respect definite optima exist; although it is difficult to determine these for the substrate of the cell sap, yet the sensitivity of micro-organisms to acidity depends largely on the chemical composition of the nutrient solution and especially on the nature of the acids present.

TABLE XLIV

The cardinal points of hydrogen-ion concentration for four plant pathogenic bacteria. (After Kotte, 1931)

<i>Species of bacterium</i>	<i>Name of the disease</i>	<i>Primary host</i>	<i>Minimum pH</i>	<i>Optimum pH</i>	<i>Maximum pH</i>
<i>Bacterium tabacum</i>	Wildfire	Tobacco	4.6-5.0	6.7-7.1	9.2-9.4
<i>Bacterium medicaginis</i> var. <i>phaseolicola</i>	Grease spot disease	Bean	5.0-5.4	6.7-7.4	8.8-9.2
<i>Pseudomonas endiviae</i>	Leaf spot disease	Endive	5.0-5.4	6.9-7.1	9.2-9.4
<i>Aplanobacter michiganense</i>	Bacterial canker	Tomato	5.0-5.4	7.5-7.7	8.8-9.2

In general, the significance of the actual acidity as a factor in resistance to spread seems to have been overrated; thus, bacteria are held to be sensitive to acid, but it is just the plant pathogenic forms that are not so (Table XLIV) and which can, therefore, live in plant cells. In their case.

it is only in extremely acid tissues that an acid protection can be conjectured; for instance, *Bacterium vesicatorium* (syn. *Bact. exitiosum*), the agent of a leaf spot known as 'canker' of tomatoes, potatoes, &c., only begins to develop at pH 5, like the pathogens in Table XLIV. The tissues of tomato seedlings and leaves yield a value of pH 6.3-6.5, green fruits pH 5.5-4, and ripening and mature fruits pH 4-4.6. Seedlings, leaves, and green fruits are, therefore, susceptible to the disease, whereas infection experiments on ripe fruits are mostly unsuccessful (Gardner and Kendrick, 1921): evidently these are passively defended by their very acid cell sap.

For plant pathogenic fungi, also, the acidity of the cell sap, in spite of many statements to the contrary is, as a rule, probably without significance, since they are even more tolerant of hydrogen-ions than bacteria. Thus, *Taphrina deformans* (peach leaf curl; Fig. 294) grows in artificial culture from pH 3-10 and even flourishes between pH 4.3-9.6, an ill-defined optimum being shown only between pH 4-5 (Mix, 1924). It is certainly possible that the sensitivity of the parasites to acidity may be slightly greater in the meagre nutriment provided by the host plasma than in laboratory media, but their possible reaction range is likely to be far more extensive than the fluctuations in the actual acidity of the cell sap during a day or a growing season, and than the disparities existing between the individual tissues within the host.

Moreover, those fungi which parasitize highly acid host tissues can assimilate limited amounts of organic acids for their own nutrition. Thus, it is true that, of the more important *Citrus*-inhabiting fungi, *Alternaria citri* and *Diplodia natalensis* (black rot), *Phomopsis citri* and *Sclerotinia Libertiana* (storage rots), and *Penicillium stoloniferum* and *P. digitatum* (secondary on crushed tissues, &c.), none is capable of subsistence with citric acid as the sole source of carbon, although *Penicillium stoloniferum* and, to some extent, *Sclerotinia Libertiana* can do so if they have previously acquired sufficient vitality on sugar-containing substrates. On the other hand, *Alternaria citri* and *Diplodia natalensis* can utilize citric acid to some extent under favourable pH conditions, but only if a small quantity of sugar is also available; this, however, is no doubt usually the case. The acid content of the host tissues plays, therefore, a minor role in the specialization of these parasites and in the varying susceptibility of the different *Citrus* species (Camp, 1923).

6. *The Osmotic Relations of the Cell Sap as a Factor in Resistance to Spread*

The suction pressure of the pathogen must be greater than that of the host. Such a suction pressure gradient has been actually measured at the site of infection of phanerogamous parasites (Bergdolt, 1927); thus, the ground tissue cells of *Orobanche crenata* var. *typica* (syn. *O. speciosa*), in a particular case showed a suction pressure of about 14 atm., the haustoria 12.7 atm., and the parenchyma of the invaded roots of *Vicia Faba* 8 atm. Hence, there was a suction pressure gradient from parasite to host of

nearly 5 atm. Even greater differences were observed between the haustoria of *Lathraea Squamaria* (toothwort) and the roots of *Prunus Padus* (bird cherry, Fig. 175).

Hence, a host would be osmotically protected against a pathogen if the suction pressure of its cell contents were higher than the suction pressure the parasite can employ. This eventuality, however, is scarcely likely to occur in our floristic zone. Thus, the osmotic pressure of the cell contents (cryoscopically measured) amounts to 8.3 atm. in strawberries, 17.9 atm. in apples, 10.3 atm. in sweet potatoes, and 6.3 atm. in potatoes (Hawkins, 1916). However, the maximum osmotic pressures tolerated in

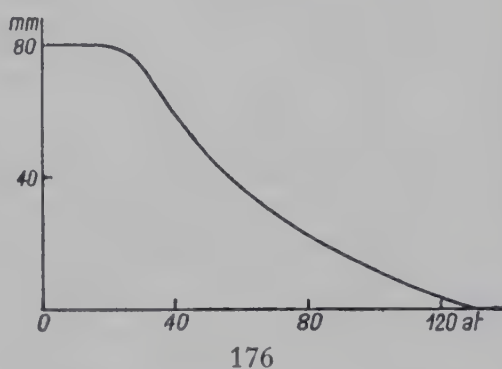
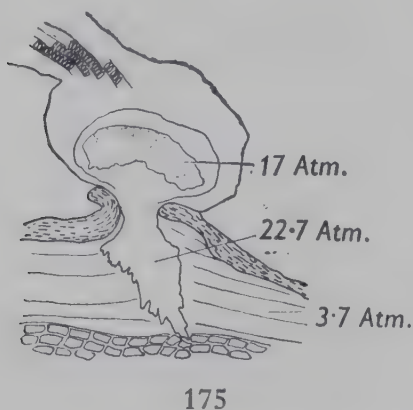


FIG. 175. The distribution of suction pressure in a haustorium of *Lathraea Squamaria* on a root of *Prunus Padus*. $\times 13$. (After Bergdolt, 1927.)

FIG. 176. The development of *Alternaria solani* in relation to the osmotic pressure of the substrate. Abscissae: osmotic pressure of the medium. Ordinates: diameter of the cultures after 10 days at 17–18° C. (After Klaus, 1943.)

glucose solutions by the fungi parasitic on these fruits are much higher, e.g. above 63 atm. in *Rhizopus nigricans* (strawberry rot), *Ophiostoma fimbriatum* (syn. *Ceratostomella fimbriata*; black rot of sweet potatoes), and *Sphaeropsis malorum* (apple branch canker and black rot), and above 39 atm. in *Fusarium radicicola* and *F. oxysporum*, the agents of a dry rot of potato tubers.

Fig. 176 shows a like tolerance in *Alternaria solani*, the agent of a dry leaf spot of potato foliage (Fig. 216) and a dry rot of the tubers: the upper limit, at which all fungal growth ceases, is only reached at 130 atm. Hence, even if it should be shown that a difference in suction pressure of the cells existed between an *Alternaria*-susceptible and an *Alternaria*-resistant potato variety, this difference would not be directly correlated with susceptibility, because the fungus can always exert a greater suction pressure than the potato cells.

7. Antibiotic Substances in the Cell Content as a Factor in Resistance to Spread

Antibiotic, germicidal substances in the cell content are protective substances (*antiseptica*) which, occurring naturally in the cells, impede the proliferation of the pathogens in the host tissues (and perhaps even their

penetration into the host). Bactericidal antibiotics are widespread in the plant world and may possibly be one of the reasons for the small number of infectious bacterial diseases of plants (p. 1). Osborn (1943), for example, established in the expressed juice of no less than 63 genera of phanero-gams, &c., belonging to 28 families, an inhibitory action, not as yet more closely defined, on two microbes pathogenic to man, *Staphylococcus aureus* (a causal agent of ulcers) and *Bacterium coli* (usually a commensal inhabitant of the bowel). Fungicidal antibiotics are much less frequent in flowering plants.

In the present section we shall discuss some antibiotics of chemically defined constitution and activity, and in the following section (8, the pre-formed antibodies) some germicidal principles of still unknown chemical constitution.

In general, plant alkaloids exert only a slight toxic action on the specific disease-agents of the relevant host groups, although, for instance, *Cladosporium fulvum*, the causal agent of leaf mould of tomatoes and other Solanaceae (Fig. 280), reacts very sensitively to solanin (Fig. 177).

On the other hand, with a non-specific parasite, *Phymatotrichum omnivorum* (p. 180), tests with 62 different alkaloids showed a certain relationship to exist between the alkaloid content of the roots of the various hosts and their resistance to the fungus (Greathouse and Rigler, 1940). The maximum toxicity was shown by sanguinarin, which completely inhibits fungal growth even at a dilution of 2.5×10^{-6} , the minimum by aconitin, solanin, &c. (or the limit of the order of parts per thousand). Comparable inhibitory values of alkaloids likewise exist in regard to other non-specific parasites of these host plants such as *Sclerotium Rolfsii* (p. 112), *Corticium vagum* (p. 179), *Armillaria mellea* (p. 44), *Verticillium albo-atrum* (p. 179), &c.

In addition to alkaloids, by analogy with the observed antiseptic properties of the phenol derivatives in human medicine, all the possible phenols, tannins, flavonols, &c., have been claimed as factors of resistance in fruits, bark, wood, and finally even in the leaf tissues, but only in rare

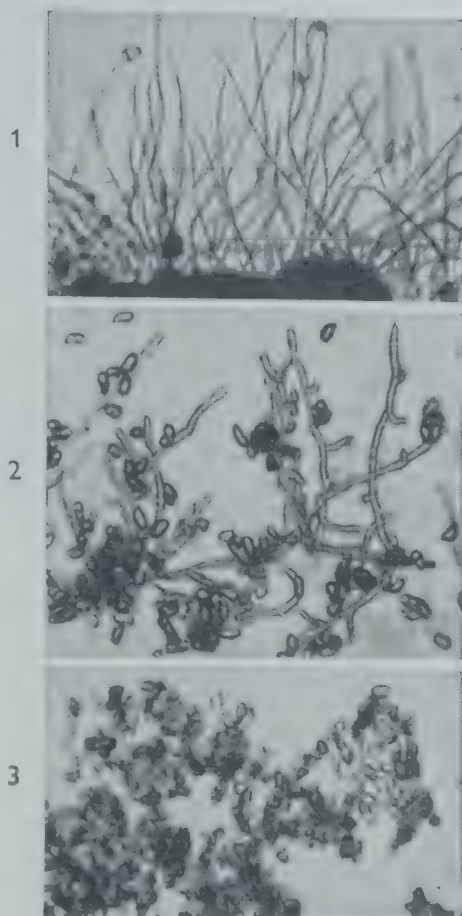


FIG. 177. The influence of solanin content on the spore germination and development of *Cladosporium fulvum* photographed after 15 hours. 1. Solanin-free (tobacco leaf decoction); natural development. 2. Low solanin content (potato seedling decoction, 1g. substance per 40 c.c. water); hyphae stunted. 3. High solanin content (4 times more concentrated than 2); no germination. Approx. $\times 400$. (After Agerberg *et al.*, 1933.)

instances has this claim been definitely confirmed. Thus, pine wood contains two fungicidal phenolic substances, pinosylvin and pinosylvinmono-methylether; a content of the latter of about 0.7–0.8% largely protects the wood against *Lentinus squamosus*, a saprophytic destroyer of felled and processed wood, but not against *Trametes radiciperda*, the butt fungus, which is the most dangerous parasite of standing pine wood (Rennerfelt, 1943), so that here the toxic action fails.

In general, however, the role of phenols and tannins as factors of resistance to spread remains merely a working hypothesis since they occur in the tissues of susceptible as well as of resistant plants. Because of their great chemical instability it is difficult to prove conclusively that they operate in the cells in just the same combination and concentration in which they are tested in the laboratory apart from the host. Even in human medicine the relations between the structure of the phenol derivatives and their antiseptic properties have been only partially elucidated.

Table XLV shows the marked differences in activity of six closely related phenols in respect of *Colletotrichum circinans* and *Botrytis allii* (two specific onion parasites), *Aspergillus niger* (a non-specific onion parasite), and *Gibberella Saubinetii*, the agent of a foot rot of wheat. Their thresholds of sensitivity are very low in comparison with the antiseptics of human medicine. In general, the position of the hydroxyl groups seems materially to influence toxicity to fungi: in the meta position (phenol, resorcin, phloroglucin), it decreases with increasing molecular weight, whereas, in the ortho position (phenol, catechol, pyrogallol), it increases.

The difficulties are further enhanced by the fact that, at high dilutions (millionths), some of these substances stimulate the parasites (cf. Newton *et al.*, 1929); where, then, do the *dosis stimulans* and the *dosis toxica* occur in the cells? Still other parasites can partially decompose tannins and utilize them as a source of carbon (like citric acid as indicated on p. 271); this applies in particular to the specific bark and wood dwellers, against which the tissues ought to be quite specially protected by their tannin content.

TABLE XLV

The growth-inhibiting thresholds of some phenol derivatives in Czapek's nutrient solution. (After Walker and Link, 1935)

Compound	<i>Colletotrichum circinans</i>	<i>Botrytis allii</i>	<i>Aspergillus niger</i>	<i>Gibberella Saubinetii</i>
Phenol C_6H_5OH . .	1:3,200	1:1,600	1:1,600	1:3,200
Catechol $C_6H_4(OH)_2$ 1:2 .	1:6,400	1:1,600	1:400	1:3,200
Resorcin $C_6H_4(OH)_2$ 1:3 .	1:800	1:200	1:400	1:400
Hydroquinone $C_6H_4(OH)_2$ 1:4	1:3,200	1:800	<1:200	1:800
Phloroglucin $C_6H_3(OH)_3$ 1:3:5	1:100	<1:100	<1:100	<1:100
Pyrogallol $C_6H_3(OH)_3$ 1:2:3	1:2,600	1:6,400	1:100	1:6,400

As an explanation, the hypothesis was advanced that the toxic phenols do not occur as such in the cell content, but are only released from complex

combinations by the fungal enzymes (auto-intoxication of the parasite; cf. Newton and Anderson, 1929). In this case it must be assumed that the susceptibility of a given host depends on enzyme inactivators which hinder the release by the fungus of the toxic phenol components; all this is certainly possible but hard to prove.

The significance of the mustard oils in general resistance to spread is clearer than that of the phenols, tannins, &c. Free allyl mustard oil is very toxic to the parasites listed in Table XLV, inhibiting them at a concentration as low as 20–40:10⁶, in which they actually occur, for instance, in the cell sap of onions (Walker *et al.*, 1937). Among the Cruciferae, too, a correlation exists between the allyl mustard oil content and resistance to club root, *Plasmodiophora brassicae* (Rochlin, 1933). Generally speaking, the toxicity of the mustard oils decreases in the order allyl, phenyl, methyl, ethyl; the action of isomers may vary greatly.

It is just this question of the significance of allyl mustard oil for the resistance of the host to spread that reveals the technical difficulties to be overcome in obtaining evidence of the nature of the toxic substances in the cell content. Allyl mustard oil occurs in plants in a glucoside combination as sinigrin; this, however, is non-toxic to the fungi under discussion, so that they can readily occupy sinigrin-containing host cells as long as the mustard oil-splitting enzyme, myrosin, does not penetrate into the cell through a wound. Mass analyses of whole tissues easily lead, therefore, to erroneous conclusions.

Finally, an interesting case of resistance to spread has been observed in the case of heteroauxin. It has always been difficult to explain why pathogens develop only at a certain distance behind the growing-point and do not attack the latter directly unless it is otherwise weakened. Sometimes, the reason seems to be that heteroauxin exerts a harmful effect on micro-organisms inhibiting, for instance, the germination and growth of *Tilletia tritici* (bunt of wheat), at a concentration as low as $4 \cdot 10^{-10}$, and *Cercospora herpotrichoides* (Fig. 92) at a concentration of $4 \cdot 10^{-6}$ (Défago, 1940). These are lower heteroauxin concentrations than have been demonstrated for plant tissues. On the other hand, another agent of foot rot, *Ophiobolus herpotrichus*, is much less sensitive to heteroauxin.

Thus, wheat seedlings only permit the two former parasites to develop by providing them with their specifically important growth substances (aneurin for *Tilletia tritici*, pyrimidin for *Cercospora herpotrichoides*) but, on the other hand, they inhibit them by another growth substance (heteroauxin) and so keep them at a distance from the vital growth zones (the growing-points).

8. Pre-formed Antibodies as a Factor in Resistance to Spread

Antibodies were originally thought to be defensive and inhibitory substances newly formed by the host in response to infection. Subsequently, however, it became apparent that certain principles which acted in the same way, and perhaps also were identical chemically, already occurred

spontaneously in organisms: the blood of rats, for example, is naturally bactericidal to anthrax bacilli. Thus, there is no essential difference between the inhibitory principles formed before and after infection. This is not surprising, since an infected organism can scarcely exhibit totally new capacities as a result of infection, but merely pursues its earlier way of life and carries on as it did when in good health, only more intensively.

However, this identity of some of the principles developed before and after infection creates certain difficulties of technique, since if inaccurate experimental methods be used, the former may simulate the latter. Further, this identity makes nomenclature awkward since it readily leads to mistakes. The principles originally existing in the cells in the absence of the pathogen, that is to say, prior to infection, are termed normal, physiological, autonomous, pre-formed, or pseudo-antibodies; they are factors of passive resistance. On the other hand, those principles newly formed after infection, in response to the attack, are termed true or induced antibodies; they are products of the active cellular defence reactions to be discussed later.

The anti-bacterial action of the antibodies is either (a) agglutinating (flaking, precipitating, crippling of the flagella), or (b) bactericidal (killing), or (c) lytic (dissolving), and their anti-fungal action (d) is generally inhibitory to germination and growth (Carbone and Arnaudi, 1930; Chester, 1933; Baldacci, 1942). How far these effects are modes of operation of the same substance cannot at present be decided; similarly, it is only technical considerations that lead to the distinguishing of three separate anti-bacterial active principles whereas, in the case of fungi, they are all included under the one heading 'inhibition'.

(a) Pre-formed agglutinins. Since 1902 (Kobert, ricin, &c.), the agglutinating effect of plant substances on human and animal blood has been demonstrated for the most diverse objects including seeds of Leguminosae, rice and maize extracts, potato juice, &c. (haemagglutinins). Similarly the expressed sap of many plants contains natural agglutinins against all sorts of bacteria (bacterio-agglutinins). The following may be cited as examples: expressed juice of potato tubers and of leaves of *Sempervivum Hausmannii* against saprophytes like *Bacillus vulgatus*, *Bacterium putidum*, and *Bacillus asteroides* (Wagner, 1915); expressed juice of *Cotyledon Scheideckeri*, a species of Crassulaceae related to the houseleek, against *Bacterium typhi abdom.* and *Vibrio cholerae asiat.* (Kritschewsky, 1914, 1915); expressed juice of potato tubers, precipitated in 95% alcohol and then dissolved in distilled water and adjusted to the normal hydrogen-ion concentration of the cell sap (Berridge, 1929), against *Bacterium coli* and parasites of other plants, like *Bacterium delphinii* (black spot of larkspur), &c.

The fact that the cell sap in potato tubers always contains agglutinating principles against saprophytic bacteria helps to explain why the tubers, in spite of their rich supply of readily digestible nutrients and in spite of

their suitability when cooked to serve as a substrate for these ubiquitous bacteria, are not attacked by them in the living state even through injuries; the agglutinins, as factors of resistance to spread, generally appear to fend them off. On the other hand, the bacterial agents of wet rots, such as *Bacillus atrosepticus* (black leg) and *Bacillus carotovorus* (soft rot of carrot, also transmissible to potato), react only slightly or not at all to the agglutinins; hence, the natural protective substance fails against them and this is why they are able to live parasitically in the tuber tissues.

The agglutinative capacity of the expressed juice of one and the same plant against different bacteria, and of different plants against the same bacterium varies (dilution series!). Further, their physico-chemical properties also show divergencies; e.g. the Wagner agglutinins succumb to 2 hours' heating at 45° C, whereas Kritschewsky's are highly thermostable (134–144° C.). It is a problem, therefore, of diverse groups of substances and not only of proteins. Berridge's agglutinating principle is most efficient at the hydrogen-ion concentration of the cell sap.

(b) Pre-formed bactericidal antibodies. What is the basis of the inhospitableness of the plant organism (p. 253), which after all offers in its intercellular spaces shade, humidity, and no doubt also some nourishment? Non-specific saprophytes, e.g. *Bacterium prodigiosum* (red discoloration of starchy substrates, e.g. bread), or parasites of man, such as *Bacterium pneumoniae* (pneumonia) and *Bacterium ozaenae* (the presumed agent of foetid nasal catarrh), grow vigorously in the expressed sap of *Iris* spp., or that of *Cotyledon glauca* (syn. *Echeveria glauca*) whereas, in the intercellular spaces of these plants they perish within a few days (Söding, 1939, 1941). The reason lies in a soluble bactericidal substance, which evidently diffuses from the leaf cells into the intercellular spaces.

(c) Pre-formed lysins. The presence of lysins of animal and human blood (haemolysins) has been observed, together with haemagglutinins, in many expressed juices. The same is true of bacteriolysins; thus, bacteria-dissolving principles occur in the above-mentioned Wagner expressed juices of potato.

(d) Pre-formed fungus-inhibiting principles. In a sterile expressed juice, prepared from the relevant organs of a susceptible maize variety, the pathogen of maize smut (*Ustilago zeae*) can make good growth, whereas in a comparable expressed juice from the same organs of a resistant maize variety it develops badly and produces only about 1% of the normal growth (Ranker, 1930). Similarly, the growth of *Fusarium lini* (wilt disease of flax) is substantially reduced by the addition to the nutrient solution of the extract of a resistant plant, whilst that of a susceptible plant has no effect (Reynolds, 1931). Black rust infection of cereals is diminished by the germination of the spores in the cell sap of a resistant wheat variety, whereas that of a susceptible one exerts no inhibitory effect (Newton *et al.*, 1929).

In a good nutrient solution, supplemented by 1% expressed juice from cotton roots (i.e. a susceptible host) the very aggressive *Phymatotrichum*

omnivorum (p. 180) yields in 38 days at 25–32° C. a mycelial dry weight of 249.4 mg.; with 1% expressed juice of maize (resistant host) 0 mg.; and in the control, without expressed juice, 438.6 mg. However, in other sample pairs, e.g. carrots and onions, the difference is less marked (Ezekiel *et al.*, 1932).

In certain cases, therefore, biochemical inhibitory substances, really 'pseudo-antibodies', co-operate as factors in resistance to spread, in other cases the alkaloids, &c., discussed on page 273. It is not to be expected that the resistance to spread in all the members of a host range as extensive as that of *Phymatotrichum* (p. 180) should all depend upon identical factors.

It is common to all these pre-formed bacteria- and fungus-inhibiting principles that they do not, as a rule, prevent the pathogen from attacking but weaken it in the tissues of the host so that it succumbs more readily to the defence reactions now to be discussed.

II. THE DEFENCE REACTIONS

By a defence reaction we understand a vital process initiated in a host by a pathogenic agent and directed more or less specifically against that agent itself.

Thus, defence reactions are merely a special form of the general regulatory processes, to be discussed in the next chapter, whereby the plant organism attempts to restore the equilibrium disturbed by the infection. They appear only when the plant is a suitable host and they include the whole range of potentialities between its qualification and its 'inclination' to serve as host (p. 249). The expression 'immune reaction' is often used instead of 'defence reaction' and, although the two expressions are not entirely synonymous, any difference has been lost in current usage.

The capacity for defence reaction creates a certain grade or level of immunity in the plant body. If its defence readiness makes it proof against a given pathogen, it is immune to it. The term immunity, therefore, implies the capacity of an organism for active defence against a pathogenic agent; according to this definition the expression 'active immunity' is a pleonasm.

Defence reactions are autonomous (spontaneous) or induced (acquired) depending on their genesis. The capacity for autonomous defence reactions is present to its full extent in the organism from the beginning as an innate specific character (natural immunity of varying degrees). On the other hand, the reaction capacities involved may be dormant or latent in the plant and only awakened or activated by a specific primary infection. The plant's survival of this primary infection sensitizes its disposition to resist so that it now develops its full immunological efficiency; in this case the expression 'induced defence reactions' is used and the capacity for defence conditioned by them is termed 'acquired immunity'. Hence, the latter is restricted to definite individuals and implies a position peculiar to

the individual within the range of species variation, which derives from a special event in the individual's life-history.

According to their degree of activity defence reactions are 'normergic' or 'hyperergic'. In normergic reactions, cause and effect stand in a 'normal' or proportional relationship to each other, whereas hyperergic reactions are disproportionately exaggerated in relation to their causes.

Furthermore, the physician also speaks of immunization and immunity when the defence reactions are not exercised by the diseased organism, but when protective substances developed by another organism in the course of a similar or homologous disease and cure are introduced by clinical methods into the body to be protected (passive immunization); in both cases it is a question of an homologous protection in contrast to chemotherapy in which the protective medicaments are entirely heterologous. However, the two immunities depend on different biological conditions; in the immune reactions, *sensu stricta*, the body itself is able to form the protective substances whereas, in passive immunization, it need only be in a position to tolerate them in the introduced form and to harbour them for a certain time.

We shall, therefore, discuss the two phenomena separately. § 1: 'Defence Reactions *sensu stricta*', and § 2: 'Passive Immunization' (p. 351).

§ 1. Defence Reactions *sensu stricta*

Plant defence reactions can be arranged in three groups according to the object against which they are primarily directed (Table XLVI).

TABLE XLVI

Schematic arrangement of the defence reactions under discussion.
(After Gäumann, 1944)

<i>Object of the reaction</i>	<i>Genesis</i>	<i>Intensity</i>	<i>Type of reaction</i>
The pathogen itself (anti-infectious defence reactions)	autonomous	normergic	Plasmatic defence reactions (p. 281)
		hyperergic	Necrogenous reactions (p. 290)
The metabolic products of the pathogen (antitoxic defence reactions)	induced	normergic	Premunity (p. 307)
	autonomous	normergic	Histogenic demarcations (p. 330)
		hyperergic	Gummosis demarcations (p. 341)
Sensitivity of the organism itself (induced tolerance)	induced	—	De-sensitization (p. 343)

1. Anti-infectious defence reactions which are aimed directly at the pathogen itself and which are intended to weaken and destroy it (fixation or destruction of the germs); they are, therefore, also described as antiparasitic defence reactions.

2. Antitoxic defence reactions (p. 328) which, in the first place, are not directed against the pathogen but against its metabolic products, especially

its toxins, and further against the necrotic products of the damaged plant cells and tissues which they tend to render harmless (fixation or destruction of toxins).

3. Induced tolerance (p. 343) which is directed, contrary to the previously given definition, neither against the parasite itself nor against its metabolic products, but against the sensitivity of the host plant itself to these pathogenic agents. An adjustment, a de-sensitization, occurs in the plant so that it no longer responds to the pathogenic agent but tolerates it without manifest reaction and, consequently, does not become diseased.

These three types of reaction do not, of course, run parallel to each other or succeed one another independently in the plant body but work together, and it is only for reasons of technical presentation that, in the following sections, one of the three aspects, according to the nature of the example, is emphasized exclusively.

1. *Anti-infectional Defence Reactions*

The existence of anti-infectional defence reactions is shown by the fact that most infectious plant diseases do not spread indiscriminately through the host (general infection), but are localized (p. 68): after a certain time the infection comes to a standstill and remains spatially confined to a particular region which often is characteristic for the given disease. The reason for this cannot lie in exhaustion of the parasite, for instance through starvation, because it first begins to form reproductive bodies after its arrest and, if re-isolated, it again grows normally. It is not easy to imagine that a parasite should first be starved by the host and then reproduce more or less luxuriantly on it for weeks on end. Further, many agents of 'leaf spot diseases' which produce strictly local effects in uninjured foliage proliferate freely in the same leaves if previously these have been killed by narcotics. It cannot, therefore, be lack of food that formerly restricted them; anti-infectional defence reactions must have occurred after a given time in a certain zone around the focus of infection which precluded the further advance of the parasite.

Kunkel's observations (1918) on club root of cabbage (*Plasmodiophora brassicae*; Fig. 128) point to a similar conclusion. Only about 28% of the tumour cells contain the plasmodia of the pathogen; the remaining cells are free from them, not because they chanced to escape infection, but because they evidently keep the parasite at a distance by some kind of plasmatic defence activity. In rare cases where nearly all the cells are attacked, the disease runs an atypical course, the plasmodia remaining small and unable to stimulate the host cells to abnormal growth.

The present section will be divided into three sub-sections:

- (a) autonomous anti-infectional defence reactions with which the individual is endowed by nature;
- (b) induced anti-infectional defence reactions (p. 306), in which the capacity for fully effective reaction is only acquired through a primary infection;

(c) the mechanism of the anti-infectional defence reactions (p. 322); this will outline the little we know about their material bases.

(a) *Autonomous Anti-infectional Defence Reactions*

The instances of autonomous anti-infectional defence reactions in plants which have been closely investigated have one trait in common, viz. that, in spite of their name, they are as a rule unable to prevent infection and to keep the parasite at a distance. Infection, therefore, mostly 'takes', but the anti-infectional defence reactions continue, and, if all goes well, succeed in confining the pathogen to certain tissues and later perhaps even destroying and ejecting it together with the local focus or centre of infection. In

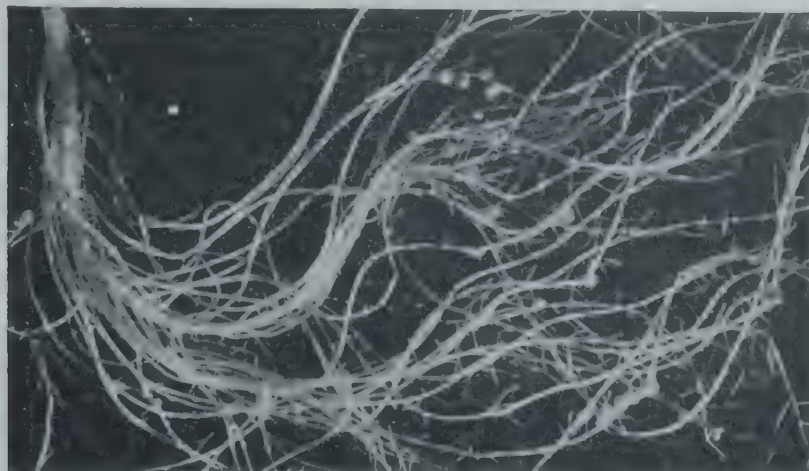


FIG. 178. Nodule formation on peas in Knop's nutrient solution, containing 1% of the normal quantity of nitrate. $\times \frac{2}{3}$. (After Gäumann, 1944.)

favourable cases, therefore, the anti-infectional defence reactions prevent the transition from infection to generalization and protect the host organism from suffering serious injury.

As an illustration of normergic anti-infectional reactions (Table XLVI) we will discuss (*aa*), the plasmatic or biochemical defence reactions and, as an instance of hyperergic anti-infectional reactions, (*bb*), the necrogenous or aborting defence reactions (p. 290).

(*aa*) *Plasmatic Defence Reactions*

The plasmatic defence reactions, as their name implies, are functions of the cell content. They may best be observed in the case of weakly pathogenic agents which remain for a long time in contact with their host and evoke chronic diseases. We shall study their effects in the phenomena of digestion exhibited *in vivo* in relation to endophytic bacteria (e.g. nodules of Leguminosae) and endophytic fungi (e.g. mycorrhiza).

First example: the localization and subsequent digestion of the nitrogen-fixing bacteria in the root nodules of Leguminosae (Fig. 178). The occurrence of infection was considered on page 16. Its further courses follow three possible lines depending on the balance of forces between parasite and host: either the host remains undisturbed by the parasite, or the parasite overcomes the host, or they are about evenly balanced.

Case 1: the host remains undisturbed by the parasite. This happens most often when the host plants possess great vitality, are suitably nourished, and, in particular, are adequately provided with nitrogen. In this case infection does not 'take' and nodules are not formed. The nodule bacteria can only gain a foothold in their host plants, i.e. the latter are only open to infection by these bacteria, when they are partially undernourished, in particular, when they are insufficiently provided with nitrogen. If there are enough nitrates at their disposal (in nutrient solutions, for instance), they can ward off the parasite, and nodule formation will not take place on account of the success of the defence reactions.

Case 2: the parasite prevails over the host either because of a diminished vitality of the host or of an enhanced vitality of the parasite.

The vitality and powers of resistance of the host are decisively reduced, for instance, if besides a shortage of nitrogen there is a deficiency of other nutrients such as boron (Brenchley and Thornton, 1925), or of assimilates (Thornton, 1930). For example, if infected seedlings of lucerne (*Medicago sativa*) are kept in the dark, their defence reactions lapse; the endophytic micro-organism gains the ascendancy, adopts a wholly parasitic way of life, ruptures the endodermis, reaches the vital central tissues, destroys the cytoplasm and the nucleus of the host cells, and, finally, penetrates into the intercellular spaces and the middle lamellae, so that the infected roots disintegrate.

Thus, the tissue disorganization which usually only occurs spontaneously with the senescence of the nodules now begins in the young seedlings in consequence of the weakening of the host and the failure of the plasmatic defence reactions. Again, therefore, there is no formation of root nodules.

On the other hand, the vitality of the parasite can be intensified, for instance, by passage through congenial hosts (Wunschik, 1925). The vitality of a strain of nodule bacteria that has existed for a long time saprophytically in field soil is initially at a low ebb; its infections seldom 'take' and nodule formation is correspondingly sparse. After two or three successive passages it becomes more aggressive, attaining equality with the host, and a state of equilibrium is established which leads to abundant nodule production. This will be considered as an ideal example in case 3. After four or more passages the parasite-host relation swings to the other extreme: the vitality of the bacterium surpasses the capacity of the host plant for resistance; thus, the equilibrium is disturbed, as it was in the lucerne experiment, through the weakening of the host. The bacteria develop abnormally large, spongy nodules at the expense of the host plant, depriving it thereby of excessive amounts of nutriment so that its growth is arrested. Thus, undue aggressiveness of the parasite, like weakening of the host, is inimical to that normal production of nodules which is advantageous to the host.

Case 3: parasite and host are more or less evenly balanced, resulting in plentiful nodule formation and nitrogen fixation. This is the agriculturally ideal case (Fred *et al.*, 1932) cited in the text-books. Two conditions are

necessary. Firstly, that the particular strain of *Bacterium radicicola* be, in fact, capable of nitrogen fixation. This, however, is not invariably the case; many strains can assimilate little or no atmospheric nitrogen but behave towards the host plants in exactly the same way as the nitrogen-fixing strains.

Secondly, that parasite and host maintain, for a certain period, an harmonious balance of forces, that is to say, that the stimulus and the reaction to it are just about equal. The vitality of the host plants is sufficiently lowered by lack of nitrogen for infection to 'take', but not lowered enough for the parasite to gain the ascendancy; in perennating Leguminosae the two partners, therefore, remain together for some years, resulting in the well-known tumour formation.

This equilibrium, now inclining to the one side and now to the other, is not a special condition in itself but represents a transitional zone between the two extremes both of which are unfavourable to nodule formation (our cases 1 and 2). In a given plant the parasite-host relationship passes ontogenetically through the following three phases.

In the first phase of the parasite-host relationship, whose initial stages we have already discussed (p. 16), the bacteria succeed in establishing themselves in the host cells (Fig. 179, 1-4) and, by way of action and re-action, achieve with them a temporary, labile equilibrium, rather like an 'armed truce'. On the one hand, they cannot overcome the resistance of the host cells, so that the infected cells remain alive; on the other hand, the host cannot eliminate the endophytic bacteria, but is, nevertheless, able to localize them in the root cortex and to prevent them from rupturing the endodermis. Hence the clinical picture becomes one of a retarded parasitism. Under the stimulus of the endophytic bacteria the familiar small tumours—the root nodules—develop from the root cortex within a few months.

In this initial phase the host plant derives no benefit from the presence of the endophytic bacteria, even if they happen to be able to assimilate nitrogen, since, during this time of strife, they do not make use of their special capacity. During this period, therefore, *Bacterium radicicola* is an ordinary gall-forming parasite. However, neither

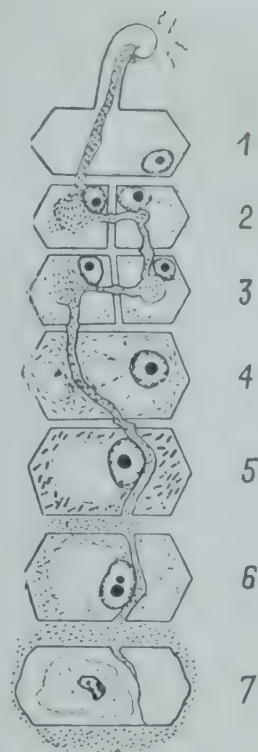


FIG. 179. Diagram of the life-history of *Bacterium radicicola* in the nodules of lucerne (*Medicago sativa*). 1 An infection thread has penetrated through a root hair. 2 and 3 Bacteria from the infection thread flood the host cells. 4 Bacteria multiply in the cytoplasm, whilst the infection thread continues to advance. 5 and 6 The intercellular bacteria are digested, but owing to its protective membrane the infection thread is not affected. The intercellular spaces and middle lamellae are infected from the infection thread. 7 Terminal stage. The cell content derived from both host and parasite is dissolved; the nodule tissues are destroyed and the intercellular bacteria return again to the soil. (After Thornton, 1930; from Gäumann, 1944.)

does the host suffer any appreciable disadvantage from the formation of the small root tumours, which is not surprising, since tumour-forming plant parasites in general are only weakly pathogenic.

The second phase of the parasite–host relationship lasts, in perennating hosts, for about 2–3 years, reckoned from the inception of the root nodules. During this period the aggressiveness of the endophyte and the resistance of the host plant are mutually adjusted and attuned (Fig. 179, 5), as in the case of the bacterial flora of the oral cavity or of the lower intestinal tract, and the bacteria are readily tolerated by their host.

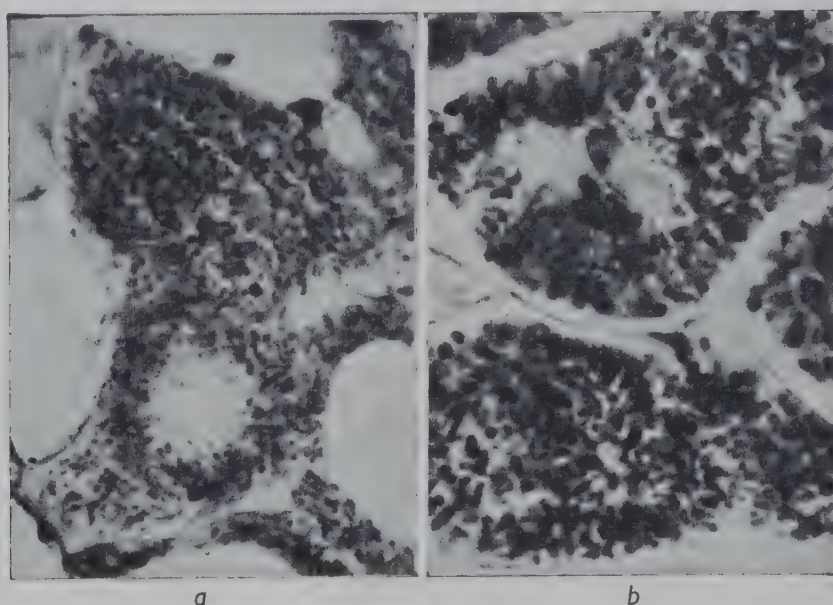


FIG. 180. Reaction forms of *Bacterium radiculicola* in the root nodules of *Neptunia oleracea*. *a* Involution forms. *b* Bacteria beginning to swell. $\times 900$. (After Schaede, 1940; from Gäumann, 1944.)

During this period of harmony many bacterial strains no longer draw all their nutrients from the host, but only their carbohydrates, &c.; in addition they assimilate the free atmospheric nitrogen and use it to form protein compounds. In this case both partners profit by their communal life, the bacterium as well as the host; the former because it obtains carbohydrates from the host plant, the latter because it obtains from the bacterium the foodstuff of which it has only a minimum. The lack of this foodstuff, nitrogen, had created the disposition to the bacterial infection. Further, the plant obtains this nitrogen from the bacteria in an organic form which, on occasion, suits it better than the inorganic nitrogen of field soil (Virtanen, 1933). This process is of great economic importance because it leads to an increased nitrogen content of the soil.

This nitrogen fixation has often given rise to unjustified doubt concerning the parasitic character of root nodule formation. It must not be overlooked that the parasite–host relationship develops in exactly the same way with bacterial strains which do not fix nitrogen; nitrogen fixation, therefore, is purely incidental, a secondary effect. In the expression ‘mutualistic symbiosis’ this eventual secondary effect is generalized.

It should be noted, moreover, that the bacteria do not fix nitrogen

spontaneously in field soil, or saprophytically in laboratory cultures, or on uncongenial hosts, or even on congenial ones in stages 1-3 of Fig. 179, but they do so only in 'parasitic captivity' on congenial hosts during stages 4 and 5 of Fig. 179. Thus, it is not a question of a voluntary activity on their part, but of a special service elicited from them by the congenial host which tolerates and shelters them. The parasitic relationship should not be regarded too crudely and narrowly; malaria is still an infectious disease even when, as a beneficial subsidiary effect, it curtails tertiary syphilis.

However, notwithstanding the apparently harmonious communal life of parasite and host there is no respite from the interplay of the forces by which they are swayed, so that, as time goes on, both partners show signs

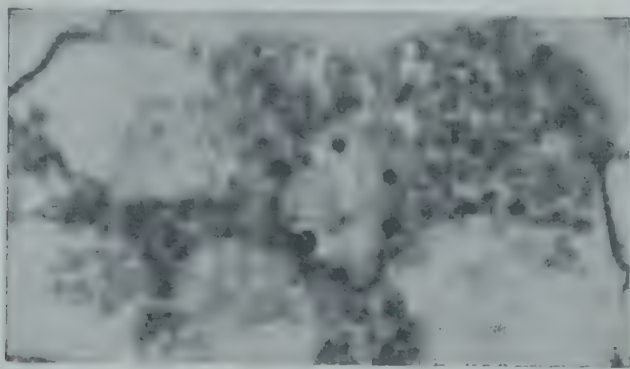


FIG. 181. Advanced stage in the digestion of swollen degenerated bacteria ('bacteroids') in the root nodules of *Lupinus albus*. $\times 1,500$.
(After Schaede, 1932; from Gäumann, 1944.)

of exhaustion. In the bacteria these manifest themselves by a tendency to curving (involution forms; Fig. 180 *a*), loss of the rod shape and of the capacity for division, so that, if isolated in culture, they make no growth; in the end they swell into vesicular bacteroids (Fig. 180 *b*). The gradual exhaustion of the host manifests itself in a loss of the power to prevent the migration of the bacteria out of the infection threads into the middle lamellae and the intercellular spaces (Fig. 179, 5-6), which become flooded with them in the region of the nodules. Since these intercellular bacteria are not subject to the influence of the plasmatic defence reactions, they do not show the above-mentioned deformations.

In the third phase of the parasite-host relationship the communal life of the two partners approaches its end in a characteristic fashion, namely, by the digestion of the intracellular bacteria (Fig. 179, 6-7). In the host cells a process of self-cleansing is initiated, carried out by means of phagocytic reactions, in the course of which lytic enzymes dissolve the bacteroids (Fig. 181). Thereby, valuable constituents of the bacteria, especially their protein compounds (provided the particular bacterial strain was nitrogen-fixing), are made available to the host so that, on balance, the latter derives from its parasite an important nutritional advantage.

Thus, between the first and third phases the parasite-host relationship is reversed, and the host now lives, if the metaphor may pass, parasitically at the expense of the intracellular bacteria, its erstwhile parasites, which it

destroys and digests to its own advantage. The intercellular bacteria, on the other hand, retain their full vitality (Fig. 179, 7), initiate the disorganization of the senescent root nodules, thereby once more reaching the soil and so closing the circle. It would be more correct, therefore, to describe the parasite-host relationship as mutualistic parasitism rather than mutualistic symbiosis.

In this third case the plasmatic defence reactions operate mainly in three directions:

- (1) they weaken the intruding bacteria, so that the latter can no longer kill and destroy the host cells;
- (2) they localize the intruding bacteria in the cortical tissues and in the tumours arising therefrom and keep them away from the conducting vessels;
- (3) they digest, i.e. they eliminate the intracellular bacteria, in which process the most varied lytic and other disintegrative operations are involved.

In the second classical example, the mycorrhiza, the parasite-host relationship is similar in character to that prevailing between leguminous plants and their nodule bacteria; this is true both of the mainly endotrophic mycorrhiza of orchids and of the mainly ectotrophic mycorrhiza of conifers and broad-leaved trees. In these cases also the plasmatic defence reactions tend to induce a weakening, localization, and elimination of the endophyte.

In orchid embryos the attachment site of the suspensor is the vulnerable area through which the hyphae penetrate. The fungi concerned need not be specific, since in most orchids numerous representatives of different systematic groups are involved.

If the invading fungus be not sufficiently aggressive, it will immediately be arrested in the first cell layers, as in case 1 of the Leguminosae. If it be too aggressive, corresponding to case 2 of the Leguminosae, it proliferates through the entire embryonic tissue and abruptly ends the life of the seedling. Where the two partners are evenly balanced (corresponding to case 3 of the Leguminosae, but valid only for a low percentage or fraction of a percentage of the seeds), the conflict between stimulus and response eventuates in a labile equilibrium: the infection advances farther than in case 1, but not so far as in case 2, and, finally, ends in a state of chronic disease which has the appearance of an harmonious balance.

In the infected tissues, therefore, similar processes take place, *mutatis mutandis*, to those in the root nodules of leguminous plants, only in special juxtaposition, instead of in temporal succession, as in the latter. Thus, the hyphae cease to grow normally in straight lines but, in every cell, are deformed by the defence reactions of the host, become entangled (Fig. 182, peripheral layer), and remain stationary for some time before attacking the next cell. It is as though they had first to overcome the resistance

of the host cell and draw new strength from its foodstuffs before they could attack the next. Thus, in the diseased cells, substances are mobilized which, like the agglutination principle, exert a crippling effect on the intruder.

As the parasite advances farther into the seedling, the senescent tangles are dissolved and digested (Fig. 182, centre). Thus, besides the agglutinative principle which arrests the parasite, a lytic, phagocytic principle gradually forms in the resistance zone of the seedling which eliminates the parasite: the seedling contrives to ward off the parasite and to prevent the most vital growth regions from becoming infected and diseased.

When the seedling has developed into a leafy plant, its rhizomes and roots are infected anew from the soil by mycorrhizal fungi (this is true both of orchids and forest trees), and in their cortical layers a conflict proceeds similar to that we have just described in orchid embryos.

At one extreme, where the host possesses great vitality, the infiltrating mycelium is completely digested and so eliminated. In orchids the host plant continues thereafter to develop fungus free. In forest trees the initially endotrophic (intramatrical) mycorrhiza are reduced by this successful defence to ectotrophic (extramatrical) mycorrhiza. Thus, at the first infection, the hyphae of *Boletus elegans* on larch roots penetrate deep into the cortical cells where, under the influence of the defence reactions of the host cells they begin to form coils; finally, they are digested and only the mainly external fungal mantle is left (Melin, 1922). Here, then, the ectotrophic mycorrhiza arise from a primary endotrophic infection by way of digestion, the successful plasmatic defence of the host against the parasite.

At the other extreme, where the host is weakened (e.g. by adverse living conditions in orchids or by an excessive lowering of the ground water-level in forest trees), the mycorrhizal fungus, on occasion, gains the upper hand, changes over to a purely parasitic way of life, breaks through the resistance zone of the host, penetrates into the roots, and may become a menace to the host.

Typical mycorrhizal formation (Fig. 184) again results only in the

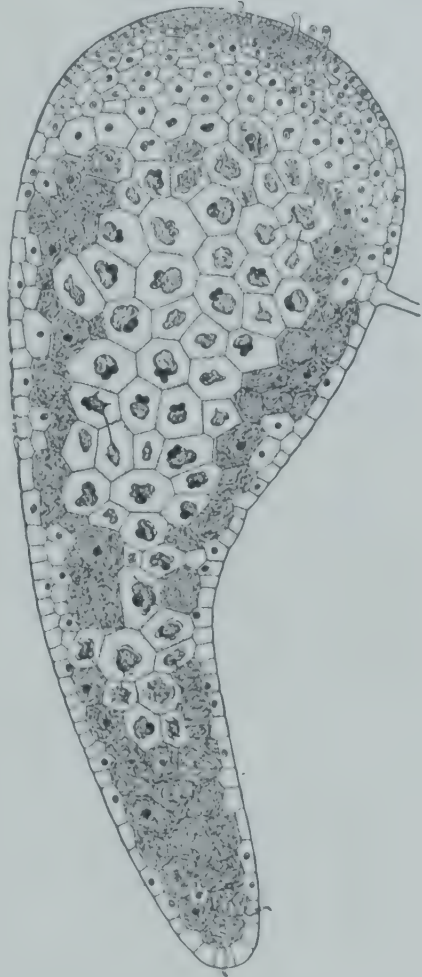


FIG. 182. Section through an embryo of *Phalaenopsis amabilis* rosea, after 50 days' infection by *Rhizoctonia mucoroides*. $\times 50$. (After Bernard, 1909; from Gäumann, 1944.)

intermediate case 3, where fungus and host plant are evenly balanced so that they continue together and remain alive. In certain cells they attain a state of temporary equilibrium (Fig. 183*a*, 'host cell'). Conversely, in other cells, e.g. those more centrally situated (Fig. 183*b*, 'digestion cells'), the parasite deteriorates with every fresh advance. If it be now re-transferred to agar, it is no longer capable of growth; its hyphal contents turn oily-yellow, lose their outlines, and disappear; the nuclei shrink slightly, becoming pycnotic and irregular in outline; finally, the hyphal walls collapse. Given

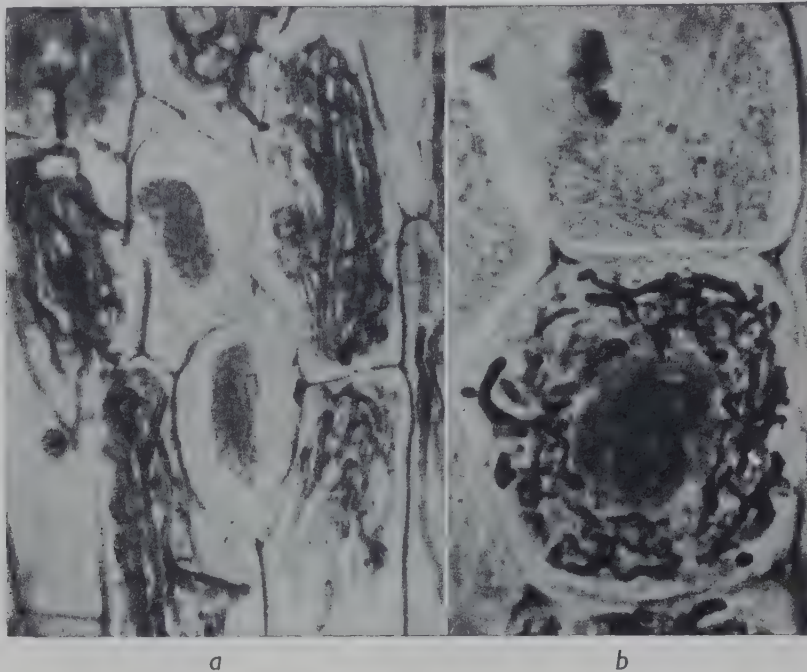


FIG. 183. The digestion of the mycorrhizal fungus in the roots of *Platanthera chlorantha*. *a* State of equilibrium between parasite and host. *b* Enzymatic destruction of the parasite: below, cell with abundant contents that are just beginning to be digested; above, nearly empty cell, contents hardly stainable, in an advanced stage of digestion. $\times 160$. (After Burgeff, 1936; from Gäumann, 1944.)

normal vitality on the part of the host, this dissolution prevents the parasite from invading the deeper lying tissues of the roots.

As in the nodules of the Leguminosae, so in the classical case of mycorrhiza, both partners derive benefit from their communal life.

In the roots of forest trees the parasitic fungus obtains carbohydrates, for which there is sharp competition in the soil, and perhaps also growth substances, &c., so that in association with congenial species of trees it develops more vigorously, under certain conditions, than as a separate unit (Melin, 1923, 1925; Björkman, 1942). On the other hand, the nitrogen balance of the tree may profit by the digestion of the hyphal coil. It is true that it is now known that the mycorrhizal fungi (in contrast to many root nodule bacteria) do not assimilate atmospheric nitrogen and, therefore, they supply the host only with the nitrogen which they have obtained by decomposing humus substances. But, where this nutrient is at a minimum, even small quantities are of great silvicultural importance and enable the

trees to establish themselves, e.g. on crude humus soils which would otherwise be unsuitable for them.

The advantage to the host seems to be even greater in orchids than in forest trees. By digesting the fungal coils, orchids profit not only from the nitrogen assimilated by the fungus from the substrate and elaborated into protein compounds (so that, under certain conditions, the nitrogen content of infected orchid seedlings is three times as great as that of uninfected seedlings; Burgeff, 1936), but (especially in the heterotrophic species) also from the carbohydrates which the fungus decomposes in the humus and absorbs. In certain cases the fungus is also the purveyor of growth substances to the host plant. Thus, the Vandaeae, a highly developed group of orchids with extremely degenerate embryos, suffer on synthetic media from an avitaminosis; they have lost the capacity for the independent production of the growth substances essential to their development and procure them from their fungal partner (Schaffstein, 1942); hence the latter is indispensable to these orchids when they are growing on a substrate deficient in growth substances. The fungus thus vicariously fulfils vital functions for the host, rather like the feeding-bottle and baby-food for the infants of mothers incapable of lactation.



FIG. 184. Ectotrophic mycorrhiza on coralloid roots of *Pinus sylvestris*. × 7. (After Fries, 1942.)

In these border-line cases where the host derives a more vital advantage than the parasite from the parasitic relationship, discussions on the 'significance of the mycorrhizal formation' readily deteriorate into arguments about words. What is pertinent to our study of parasitology is that as in the root nodules of the Leguminosae, so in the mycorrhiza, plasmatic defence reactions regulate the cohabitation of parasite and host by their crippling, localizing, and eliminating effects. We shall, later, briefly revert to these processes in the sub-section on the mechanism of the anti-infectious defence reactions.

In acute infectious diseases also, the normergic plasmatic defence reactions show the same tendencies to a weakening, localization, and elimination of the pathogen as have been described here for two chronic infections. Further, the advancing parasite doubtless evokes spontaneous plasmatic defence reactions like those just described by which, on occasion, it is suddenly, for no apparent external reason, arrested, localized, and later partially corroded and digested. However, where the disease pursues a rapid and, therefore, frequently anomalous course, only one or other of the foregoing symptoms can usually be recognised, so that the complicated relationships described above are not always present.

In the acute infectious diseases, as in the chronic, the efficiency or level of activity of the plasmatic defence reactions is not, in general, very high. It has been noted that they are mostly unable to prevent infection, one of the reasons for this being, perhaps, that their mechanism is set in motion late and operates slowly.

As a rule, however, they are able only to weaken and localize the intruding parasite but not to eliminate it. The digestion and elimination processes are restricted, in the main, to single cells and do not lead to a general self-cleansing, an auto-sterilization of the infected tissues, that is, to a general healing; the infected tissues usually remain permanently diseased. Infectious plant diseases, therefore, are mostly incurable and their harmful effects usually irreparable.

The plasmatic defence reactions are, however, far from negligible. With a favourable course they retard or prevent the transition from local infection to generalization: they weaken the pathogen, localize the contagion in a (usually chronic) focal disease (in contrast to a systemic one), and thereby preserve the host from a lethal outcome of the infection. They do not, however, eliminate the pathogen, and they do not usually cure the infection or heal the diseased tissues.

(bb) *Necrogenous or Aborting Defence Reactions*

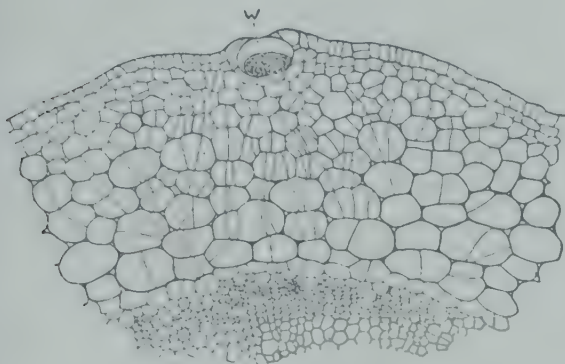
The defensive potentialities of plants are not exhausted with the failure of the normergic plasmatic defence reactions; in many infectious plant diseases new hyperergic reactions of another character now come into operation, and these will be considered together as a group, the necrogenous or aborting defence reactions. In this connexion we understand by a necrosis, the death of local cells or tissues resulting from the excretion of the toxic products of metabolism and decomposition.

The character of necrogenous defence reactions is demonstrated most clearly in an extreme case, that of the reaction of certain potato varieties to infection by *Synchytrium endobioticum* (wart disease of potato; Fig. 187). When the myxamoebae have reached an appropriate tissue (in the tubers, the eyes or their immediate vicinity) they lose their flagella, surround themselves with a membrane, dissolve a small pore in the outer epidermal wall (there is no cuticle), and release their protoplasts into the interior of the cell: the infection 'takes'.

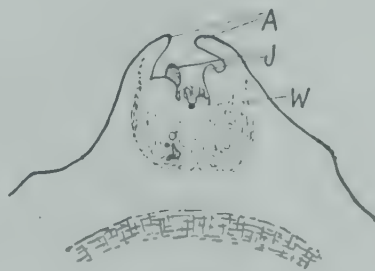
This process is enacted with the same frequency and in the same manner in both the wart-susceptible and the wart-immune potato varieties; hence, the latter, unlike *Berberis Thunbergii*, &c. (p. 250), are not endowed with any resistance to penetration. In both types, moreover, the normergic plasmatic defence reactions do not suffice to exclude the infection; both types are susceptible to infection. It is only the subsequent fate of the invading parasite that is different.

In the wart-susceptible varieties, parasite and host appear to tolerate each other. The infected cells do not show any visible injury and, at first, there is no clear-cut reaction on the host's part. It is evident that patho-

logical disturbances occur in the infected cells, manifested, *inter alia*, by an altered structure of the plasm and, no doubt, we may also assume that plasmatic defence reactions operate, but they are not evident as such and do not lead to the elimination of the infection. Parasite and host are, as it were, congenial or adapted to one another; they are in fact at variance, but they tolerate each other. They remain in contact and together enter on a parasitic communal life eventuating in disease, a communal life similar in its conditions to that existing between the bacteria and the roots in root nodules of leguminous plants (p. 282). A communal life of this kind, resting on a certain tolerance between parasite and host, is termed eusymbiosis.



185



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FIG. 185. Wart-susceptible potato, var. Deodora. Numerous cell divisions are occurring around the hypertrophied host cell *W*. Approx. $\times 50$. (After Köhler, 1928; from Gäumann, 1944.)

FIG. 186. Young wart on a shoot axis of a potato, var. Industry. Explanation in text. $\times 18$. (After Köhler, 1928.)

In these eusymbiotic potato-*Synchytrium* pairs, therefore, the infection remains in existence and subsequently initiates that stimulation of adjacent tissues which leads to the gall reaction; the neighbouring epidermal cells expand to form a circle which is later incorporated in the wart 'rosette'. Moreover, under the influence of the developing parasite, new cell walls are formed at random in the deeper surrounding tissues (Fig. 185). Thus, the wart tumours do not, for the most part, consist of infected cells, but they are induced in the non-infected neighbouring tissues by the infected cells.

In weakly susceptible varieties the gall reaction is slight; it results only in insignificant circular swellings and not in the typical cauliflower-like excrescences, so that economically the damage is small. On the other hand, in highly susceptible varieties the infection causes a very extensive malformation of buds and shoots, giving the familiar warty growths (Fig. 187) in tubers, stolons, and the base of the leaf stalks; with it there is a corresponding loss in yield. Fig. 186 shows a diagrammatic, median longitudinal section of a young wart: the centre of the wart is a rosette-like formation around the host cell (*W*), then come the inner protuberance

(\mathcal{F}), and the outer (A) representing two rings of deformed foliage primordia. The whole structure becomes coralloid, being forced up by the secondary meristem, and branches irregularly.

In the immune varieties the situation is different. Here the parasite and host are incompatible, their communal life is parabiogenic: they react antagon-



FIG. 187. Two warted tubers of var. Deodora. Tuber on the left in longitudinal section; tuber on the right in transverse section. V proliferation arising from scale bases. K a proliferation from a bud in which the individual leaves, 1, 2, and 3, can still be distinguished. $\times \frac{4}{5}$. (After Köhler, 1925.)

istically to one another and so, in addition to the normergic reactions (and especially if these fail) 'emergency' hyperergic reactions develop at the focus of infection, which are of such intensity that they can be observed microscopically.

In certain varieties, e.g. in 'Ackersegen', the reactions are seen in the infected epidermal cells and lead to their necrosis; the cells die within a few hours or days and their protoplasm is converted into a peculiar, strongly chromogenic, alveolar mass (gummosis), they lose their turgescence and are compressed by neighbouring cells (Fig. 188).

This aborting reaction of the infected epidermal cells has a twofold

consequence for the invading parasite. On the one hand it is injured by the necrotic products; on the other hand it is cut off from its livelihood since being an obligate biotrophic parasite it can feed only on living plant tissues. Therefore, when the host cell responds to the infection by dying, as if from shock or collapse, the parasite dies with it and the infection becomes abortive. Hence, the immunity of the variety 'Ackersegen'

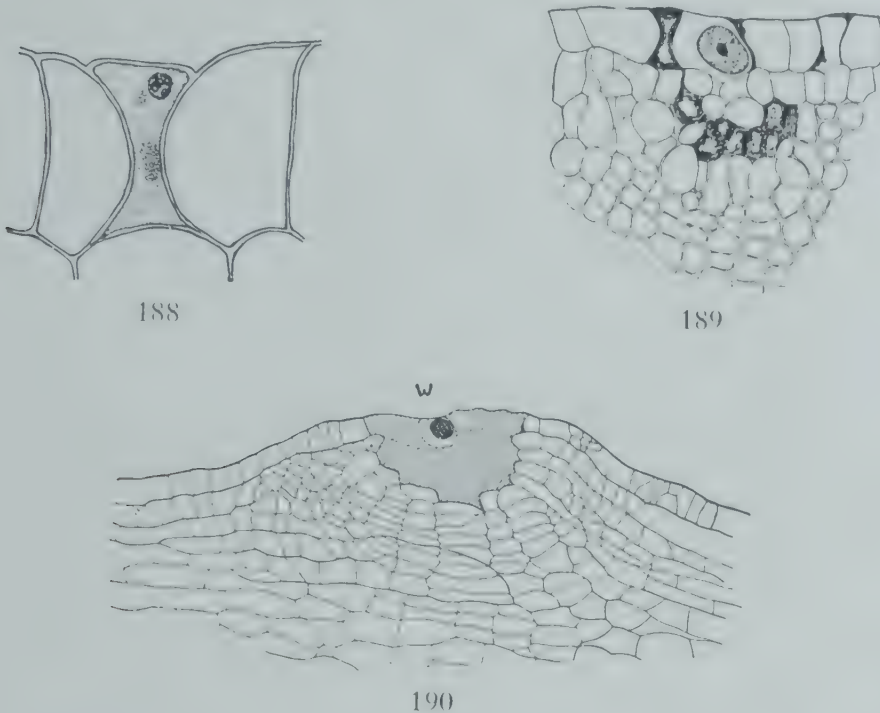


FIG. 188. Hyperergic necrotic reaction of a wart-infected epidermal cell of a potato, var. Ackersegen, a few hours after infection. The protoplasts of the parasite, darkly shaded, are still intact. $\times 825$. (After Köhler, 1931.)

FIG. 189. Early stage in the insulation of an infection locus by a 'necrotic halo', a zone of necrotic cells, in the potato, var. Paulsens Juli. $\times 250$. (After Köhler, 1928; from Gäumann, 1944.)

FIG. 190. An aborting zone around an infection centre in a wart resistant variety, about two weeks after infection. *W* hypertrophied host cell. The necrotic tissue is shaded. $\times 70$. (After Köhler, 1928; from Gäumann, 1944.)

depends on the fact that the infected cells simply cannot tolerate the parasite and so perish together with it.

In certain other immune potato varieties, such as 'Prussian', the epidermal cell directly attacked remains at first unaltered; but, stimulated by substances diffusing from the parasitized cell, hyperergic necrogenous reactions are set up in the non-infected neighbouring cells (Fig. 185) and produce in them a general gummosis (Figs. 189, 190). In consequence of the necrotic products and the interrupted supply of nutrients, the local focus of infection, together with the parasite, is destroyed within 2-3 weeks; it becomes encapsulated, isolated as a necrotic spot, and the parasite is prevented from maturing and reproducing itself (sub-infection) so that the infection chain is broken and secondary infection does not take place.

Medically, in such cases, the focus of infection would be described as being surrounded by a local reaction zone, a focus of inflammation. Doubtless, even in the potato, the abortive focus of infection sets up a thermal reaction, but this has not yet been measured. Only of fully developed warts is it known that they are some hundredths of a degree warmer than the neighbouring healthy tissues.

In extreme cases in such potato varieties there is no infection at all because, on account of the intolerance of the host, all the parasites are destroyed together with the invaded host cells. In other varieties not all the cells react with this intensity, so that not all the invading protoplasts perish but a larger or smaller portion are preserved from destruction and

can reproduce and so carry on the infection. Hence, these varieties are only partially immune.

The very obvious necrotic processes are, of course, only the end stage of the anti-infectious defence reactions; between the moment of infection and the final débâcle, a whole chain of pathological events occurs in the cells which still need to be investigated in detail. What is important for our present consideration is that although

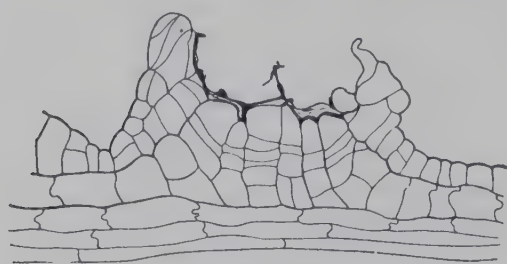


FIG. 191. The elimination of a localized wart infection in the potato, var. *Industry*. $\times 66$. (After Köhler, 1928; from Gäumann, 1944.)

in the immune varieties the infection itself succeeds, nevertheless, it does not lead to disease because the invading parasite is destroyed immediately upon entry.

These local defence reactions, moreover, are supported by the auxiliary reactions of the non-infected neighbouring tissues; here, during the production of the necrotic areas, there can occur cell divisions, with walls periclinal to the dying focus of injury (Fig. 185), which are evidently evoked by necro-hormones set free by the wound tissue. In this way a corky abstriction girdle or secondary periderm is formed, which extrudes the infection pustule (extrusion and scarring reaction) and leads to healing, a self-cleansing of the infected tissues (Fig. 191).

In wart-susceptible potato varieties the situation is, therefore, as follows. All varieties are susceptible to infection by the pathogen and, in all varieties, the factors for resistance and normergic defence reactions break down; hence, the pathogen, in all cases, penetrates into the epidermal cells and 'takes' in them. However, the post-infectious reaction of the infected cells and of their neighbours is different.

Furthermore, in one group of susceptible varieties the tissues are capable solely of normergic defence reactions; these are visible only under the microscope and are not adequate to destroy the parasite. Hence, after infection, disease follows; these varieties are susceptible to wart disease. The amount of the disease they show is proportionate to their varying disposition to gall reaction.

In another group of susceptible varieties the invaded cells, as well as their neighbours, are able to respond not only with normergic but also with necrogenous hyperergic reactions; on infection they collapse and perish but with them the infecting organism also dies and, sometime later, is in part, ejected. These varieties therefore, in spite of their susceptibility to infection, remain immune in the field.

The outcome, whether or not the potato plants become warted, depends, therefore, upon the necrogenous or aborting defence reactions. These are recognizable by the following characteristics:

1. They accompany the normergic defence reactions but only become fully active when these have failed. They require, therefore, a certain latent or preparatory period which occupies from a few hours to a few days according to the variety and to external circumstances.

In other plant diseases the period may be so short that the parasite is annihilated by the hyperergic reactions even upon its first contact with the host, so that no infection takes place. For example, in powdery mildew (*Erysiphe Martii*) of red clover (*Trifolium pratense*) the first stages of an infection, in susceptible and immune varieties alike, are similar; the oidia germinate, form an appressorium, and, like *Botrytis*, push an infection tube into an epidermal cell. In the susceptible varieties the infection tube is transformed into an haustorium and the infection 'takes' (Fig. 192 *A*). In the immune varieties the invaded cell immediately shows a vigorous antagonistic reaction, its protoplasm becomes deeply coloured and partially disorganized (sometimes even before the infection tube has entered the cell), and, finally, the whole cell collapses and with it the infection tube (Fig. 192 *B*), so that the infection aborts. In these varieties, therefore, in contrast to the *Synchytrium* example, no infection occurs.

2. Necrogenous defence reactions tend to be mainly post-infectious because of the existence of the latent period. Therefore, they do not protect the given individual against potato wart as an infection or as a pathogen, but only against wart as a disease; the parasite indeed penetrates but cannot gain a foothold, it is unable to induce any disease nor can it reproduce itself.

3. Necrogenous defence reactions are hyperergic in character. In them, cause and effect bear no direct relation to one another, the reaction is exaggerated and depends upon an idiosyncrasy of the two partners; it is,

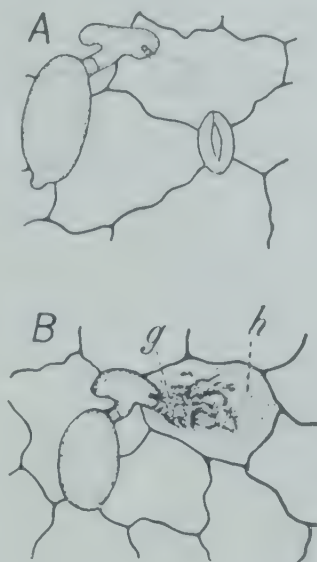


FIG. 192. Infection of *Trifolium pratense* by *Erysiphe Martii*. *A* a susceptible variety of clover, in which the parasite can form an haustorium without causing visible disorganization. *B* a mildew-resistant variety, in which the invaded epidermal cells degenerate necrotically and so destroy the parasite. *g* infection tube. *h* necrotic host cell. $\times 385$. (After O. F. Smith, 1938.)

therefore, described as an incompatible, highly susceptible, or hypersensitive reaction.

4. The capacity for hyperergic reaction is entirely innate. This distinguishes the necrogenous reactions of plants from the allergic reactions of man (e.g. anaphylaxis) with which they share their intensity of reaction. In allergic reactions the preparedness to react is not autonomous but must be evoked by a prior specific sensitization.

5. Hyperergic shock annihilates the parasite immediately upon entry; hence, in the examples cited, only a microscopically small or, at the most, a punctiform lesion occurs (point infection).

6. The effective range of a necrogenous defence reaction is limited to the focus of infection. Neighbouring tissues undergo no increase in their defence readiness and, therefore, they are not protected against homologous reinfections. Hence, the defence reaction is strictly local and does not lead to any sensitization of the plant as a whole.

7. Necrogenous defence reactions are not primarily directed against the parasite which releases them; in this, they contrast with the induced anit-infectious reactions to be discussed later. Hence, unlike the latter they are not directed towards a definite goal (*zielgerichtet*). The primary situation is rather that, in this crisis, the given cells or tissues react to the parasite in an anomalous and pathological way and perish rapidly at the site of infection. Thereby, they injure themselves first, and the simultaneous death of the parasite is an indirect and secondary effect brought about by the necrotic products to which the parasite is susceptible, which are set free at the same time.

Actually, therefore, necrogenous reactions are not true defence reactions although they have a defensive effect against obligate biotrophic pathogens. But only against obligate biotrophic pathogens; for instance, if a potato plant reacts hypersensitively to an infection by *Botrytis cinerea* (facultative biotrophic parasite or perthophyte, p. 58) and its cells round about the site of infection die suddenly, the spread of the parasite is favoured because it is not affected by the necrotic substances and grows even better on dead than on living tissues. However, when the potato plant reacts in the same way to an infection by *Synchytrium* (obligate biotrophic parasite) it inhibits the development of the fungus and itself remains healthy ('necrogenous defence reaction'). The same hypersensitiveness which facilitates infection by the vigorous *Botrytis cinerea* is lethal to the sensitive organism of wart disease. 'Subjectively', the plant is equally highly susceptible to both parasites and reacts to both in the same way; 'objectively', as a secondary effect, its intolerance saves it from disease only in the second case. Paradoxically, it remains infection free because it is hyper-susceptible.

Here we have a fresh example of life overriding logic. The case is rather like the problem of just punishment: should an offender be punished according to the measure of his intentions or according to the amount of damage perpetrated? The decision will be different according to whether the judgement be based on ethical or on practical grounds.

8. The individuals in question are not wart-resistant but wart-immune. The facts described above do not fit the conception of 'resistance'. An organism is resistant if it throws off an infection without suffering any definite injury. The conception of resistance was adopted for eusymbiotic parasite host pairs, in which the host defends itself against the parasite only by means of the normergic reactions discussed earlier. Hence, a host can be truly resistant only against eusymbiotic parasites (true immunity; left limb of the curve in Fig. 193).

On the other hand, in wart-free potato varieties, it is not the 'healthy' normergic defence reactions which determine the course of the parasitic

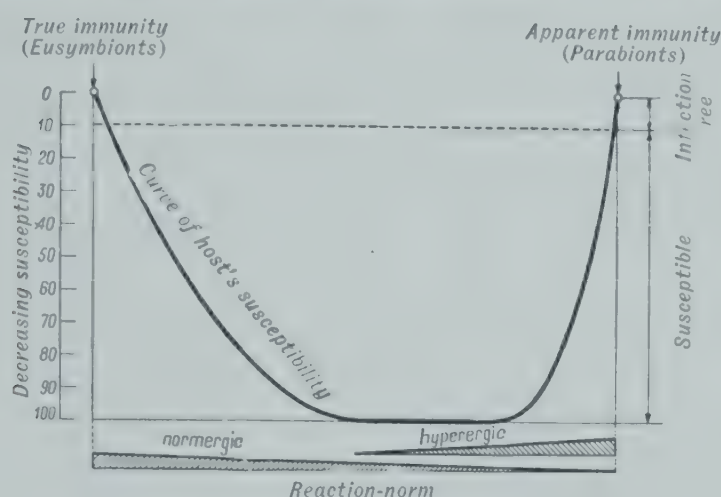


FIG. 193. The susceptibility of the host as a function of its reaction-norm. Explanation in text. (After Gäumann, 1944.)

relationship. Here its character depends upon the incompatibility of the two partners; infection is broken off and the host remains free because it is too susceptible, because its cells react over-sensitively and perish at the site of infection destroying the parasites with themselves. In eusymbiotic partners a higher susceptibility of the host leads to more severe infection whereas, in parabioc partners, the highest susceptibility results in over-sensitivity and hyperergic necrosis and, hence, in freedom from infection (apparent immunity; right limb of the curve in Fig. 193).

Fig. 193 is an attempt to illustrate these facts diagrammatically. The abscissae represent the reaction-norm; the intensity of reaction of normergic reactions decreases from left to right and susceptibility correspondingly increases. Finally, hyperergic reactions set in and increasingly outweigh the normergic action. Susceptibility is at first high but, as soon as the threshold of hypersensitivity is passed, the course of the parasitic relationship is reversed and the infection becomes abortive. The two extremes remain infection free, i.e. the strongly normergically reacting and the strongly hyperergically reacting individuals; the intermediate types are more or less susceptible.

Since the apparently immune hosts have their hypersensitivity to thank for their freedom from infection, they cannot be described as resistant;

hence they are called disease-immune; in this case, wart-immune. In colloquial speech the expression 'field immunity' is also used.

In the reaction type exemplified by potato wart disease the infection is of the 'all-or-none' kind. Either the tissue complex is able to respond hyperergically, in which case it dies and the infection is eliminated, or it only reacts normergically, in which case it remains alive together with its uninvited guest and disease breaks out.

In a second and third type of necrogenous reaction the relations are somewhat different. In the second type (e.g. potato late blight) the susceptible and immune varieties are alike capable of normergic and hyperergic reactions; however, the speed of the reaction differs and is decisive for the outcome. In the third type (e.g. rust of cereals) not all the cells in the parasite's sphere of influence are able to react hyperergically but, depending upon variety and circumstance, many or fewer cells (or only certain cells) react hyperergically, so that the disease is milder or more severe, localized or more extensive.

Second type: late blight (downy mildew) of potatoes caused by *Phytophthora infestans*. The foliage and tubers of all potato varieties are susceptible to infection by this pathogen. Infection thus 'takes' as in wart disease but, in contrast to wart disease, it is followed by a similar disease process in both *Phytophthora*-susceptible and *Phytophthora*-immune varieties. According to Müller *et al.* (1939), five phases can be distinguished in this process.

Phase I. Actual infection process. No change in the host cells can be seen under the microscope.

Phase II. The protoplasm becomes granular but has not plasmolysed; evidently normergic plasmatic defence reactions have begun. The nucleus begins to enlarge but it can still divide, i.e. it is still functional. Where the (intercellular) hyphae touch the cell walls, the latter become irreversibly stained. The parasite begins to form sporangia in abundance and is in optimum condition.

Phase III. The protoplasm becomes fibrillar, turns brown, and becomes irreversibly stained; furthermore, the cell walls in contact with the parasite turn brown. The nucleus enlarges further but the turgor of the cell is still normal. In spite of these extensive reactions of the host cells the parasite remains healthy and forms abundant reproductive bodies.

Phase IV. The reaction of the host plant (after the normergic reactions have ceased) assumes a markedly hyperergic character and both host and parasite are injured. In some nuclei shrinkage occurs. The cell walls, as a result of the infiltration of tannin-like substances from the protoplasm, begin to turn brown throughout; correspondingly the tannin reaction of the cell contents becomes progressively weaker. Many cells have already collapsed and water loss of the diseased tissues has increased considerably. In consequence of the necrogenous reactions of the host, the fungus can no longer reproduce itself; many hyphae undergo 'fatty degeneration' and if transferred to a new substrate can be induced to grow again only with difficulty.

Phase V. General necrosis. Extensive cell areas have died and become filled with an homogeneous brown substance, and the vegetative stages of the parasite have perished with them.

This sequence of reactions is characterized by the fact that, in phases II and III, the parasite and host attain a certain, although continually worsening, state of equilibrium; in the host cells plasmatic defence reactions have indeed been initiated but the parasite grows in spite of them, remains healthy, and sporulates freely. Thus, on account of the failure of the normergic defence reactions, the fungus gradually becomes dominant and over-stimulates the host tissues, so that they react hyperergically (phases IV and V). The necrogenous reactions injure both host and parasite

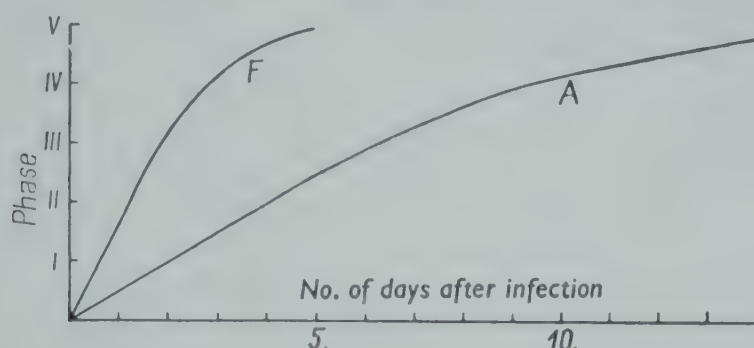


FIG. 194. The reaction of the host tissues in a *Phytophthora*-susceptible (Curve A) and a *Phytophthora*-resistant potato variety (Curve F). Explanation in text. (After Müller *et al.*, 1939; from Gäumann, 1944.)

(e.g. prevent sporulation; hence the fungus can reproduce only during phases II and III) and, finally, bring about the death of both partners.

It is, therefore, not the exhaustion of the substrate that is responsible for the degeneration of the *Phytophthora*, but the necrogenous reactions of the host; these bring about the protection of the whole plant against *P. infestans* after the normergic defence reactions have failed. The nature of the necrogenous substances is not yet known; they are, however, non-specific and are also effective against *Fusarium caeruleum* (dry rot of potato), and *F. solani* and, moreover, against saprophytes like *Penicillium 'glaucum'*.

This sequence of reactions from I to V takes place in the same way in both *Phytophthora*-susceptible and *Phytophthora*-immune potato varieties. Hence there is no fundamental difference between them; both pass through the same phases of disease and reach the same end, viz. a local necrosis. The only difference is the tempo of the course of the reaction and the time of appearance of necrosis.

Curve A of Fig. 194 represents the course of the reaction in a *Phytophthora*-susceptible variety. It proceeds slowly and, under the experimental conditions, takes about 14 days to reach the final necrosis.

This slow course has a special significance for the parasite. The disease phases II and III, which permit it a favourable reproductive period, will take about 3–7 days from infection. As *P. infestans*, under optimal temperature, begins to reproduce itself in 3 days, it can make full use of the period between the third and seventh days for the production of sporangia

and thus propagate the disease. After this the hyperergic reaction of the invaded tissues sets in, with a crippling effect on hyphae in their vicinity. But, in the meantime, other hyphae have grown farther and passed out of the necrotic area, so that the focus of infection is maintained. On the leaves it consists of dead and dying tissues (phases V and IV; Fig. 195, upper part), surrounded by a light green halo of phases III and II which, in the early morning, bears the conidiophores of the fungus on the under surface (white zone in Fig. 195), and farthest out and scarcely coloured, a narrow band of phase I, the zone into which the hyphae are only just beginning to penetrate.

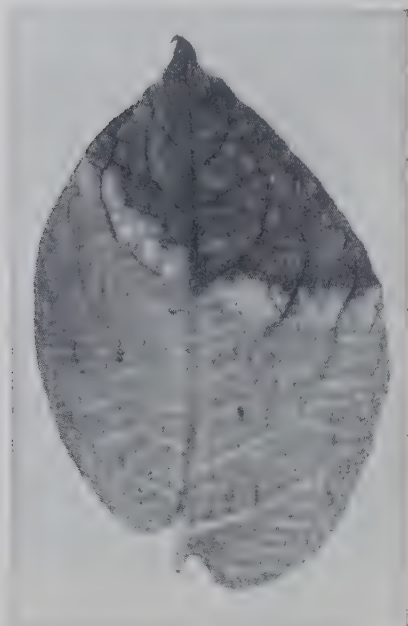


FIG. 195. A potato leaflet showing at its tip the necrotic tissue from a *Phytophthora* infection, separated from the healthy portion of the leaflet by a zone of conidiophores. Approx. $\times 2$. (After Maag, 1944.)

The reaction of the invaded host tissues occurs at a slow enough rate in variety *A* to enable the parasite to reproduce itself before the onset of the necrogenous phase and, moreover, to grow out again beyond the necrosed areas of the host. The race between the growth of the parasite and the reaction of the host eventuates, therefore, to the disadvantage of the plant. Behind the rapidly advancing parasite the host tissues are continually perishing hyperergically, but the reaction is not able to catch up with the parasite, hence the infecting organism extends ever farther and begins to shed its spores. Potato variety *A* is, therefore, susceptible to the disease.

The ideal solution, the creation of *Phytophthora*-free potato varieties, would be to breed for immunity on the basis of the normergic reactions of phases II and III. However, this

approach would not lead to a successful issue within a reasonable period since all potato varieties and related *Solanum* spp. are susceptible to *Phytophthora* and, therefore, no truly resistant material is available for crossing.

The potato breeder must, for the time being, turn in the opposite direction and look for over-sensitive varieties in which the reaction phases I–V are passed through more quickly than the time needed for the parasite to reproduce itself. This aim has been achieved in the new *Phytophthora*-immune variety *F* (Fig. 194). This is equally susceptible to both *Phytophthora* infection and disease and passes through the same reaction phases as variety *A*, but runs through them more quickly and completes phase III in 36–48 hours instead of 7 days, that is, in a time in which variety *A* still shows hardly any pathological changes.

This rapid course is again of special significance to the parasite. The latter develops only with difficulty in variety *F*, penetrates the host tissues merely to a depth of 20–30 cell layers, and then stops growing. The step

from infection to generalization is prevented, so that the sites of infection can scarcely be seen with the naked eye (spot infection). Correspondingly, the parasite does not succeed in reproducing itself and the infection chain is broken at the stage of primary infection; the parasite does not have a chance to reproduce itself because of the speed of defence reactions and the early production of necrogenous fungicidal substances. Potato variety *F* is therefore *Phytophthora*-immune.

In reality, things are not quite so simple. The speed of reaction may differ in the foliage and in the tubers so that the two organs show different grades of susceptibility (p. 248).

Thus, in Fig. 194, the space between curves *A* and *F* is taken up by intermediate potato varieties more or less susceptible to *Phytophthora*,

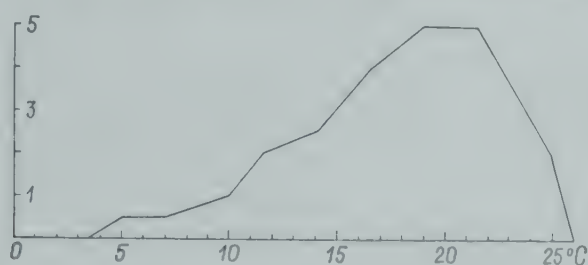


FIG. 196. The influence of temperature on the rate of development of *Phytophthora infestans*, measured in arbitrary units (1-5). After Müller & Griesinger, 1942.)

varieties in which the parasite and host approximate in their speeds of action and reaction. In these varieties, therefore, the severity or mildness of the disease is also conditioned by yet other factors and not only by the time factor of the reaction processes I-V, for example, by resistance factors such as the varying resistance to perforation of the cell wall, &c. The interpretation of the defensive behaviour in such a case is difficult.

Finally, Fig. 194 is valid only for a given external temperature, viz. that in the region of the optimum for fungus growth (about 19°C.). At this temperature the fungus is able to grow beyond the reacting tissues in susceptible varieties and thereby to escape the necrogenous effects. If the temperature be reduced to 3-4°C. the fungus, unlike *Gloeosporium album* (Fig. 162), almost ceases to grow (Fig. 196) without the reactive capacity of the host tissues being correspondingly diminished. Now, the parasite meets the same fate in the field-susceptible varieties (at low temperatures) as it does in the field-immune varieties (at optimal temperatures); the reactions of the potato tissues pursue and catch up with the fungus and the infection is halted. This is the biological basis of cold storage of potatoes in winter (in cellars, clamps, &c.). However, if the temperature rises to 25°C. for any length of time, the defensive power of the host is markedly reduced; this is one of the difficulties of potato production in the tropics.

Third type: Rust diseases of cereals. We deal next with the behaviour of a very susceptible and hypersensitive (and therefore disease-immune) variety of wheat versus biotype 11 of *Puccinia triticina* (brown rust of wheat).

The variety Little Club, of *Triticum compactum*, is highly susceptible in a eusymbiotic sense to this biotype. The appressoria of the uredospore germ tubes develop optimally on the leaves (Fig. 197, 1, *A*) and in 82% of cases the infection hypha penetrates the stomatal opening, where lateral branches from the strong sub-stomatal swelling (*B*) push between the cells of the mesophyll and form large haustoria which may attain a length of 50μ ; the fungus spreads rapidly in all directions (Fig. 197, 3 & 4) and later sporulates abundantly. The parasite is thus scarcely hindered by the

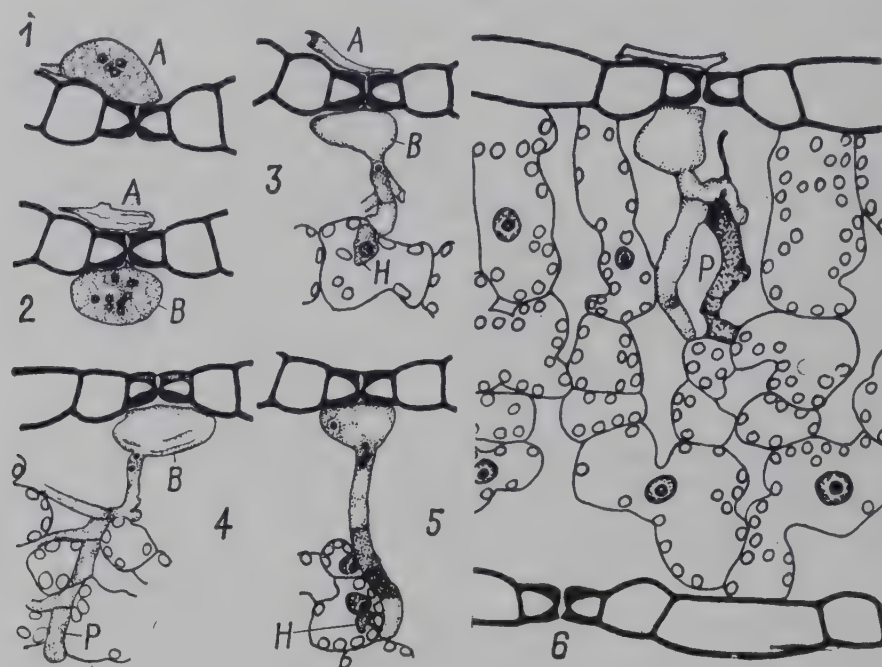


FIG. 197. The penetration of germ tubes of the uredospores of brown rust (*Puccinia triticina*), biotype 11, in a susceptible (1-4) and a hyper-sensitive wheat variety (5-6). *A* appressorium. *B* sub-stomatal swelling. *P* fungal hyphae. *H* haustoria, aborting in 5 and 6. Approx. $\times 365$. (Slightly diagrammatic, after Allen, 1926, 1927; from Gäumann, 1944.)

normergic defence reactions of the host which is, therefore, very susceptible to it. In the reverse direction the host is little sensitive to secretions and other activities of the parasite, it reacts late and then only slightly. Its cells, with the exception of the walls of the guard cells, undergo a minimal amount of injury. Even the cells harbouring the haustoria show nothing in 6 days; they neither plasmolyse nor collapse. The only visible disturbance is in the nucleus which moves towards the haustorium and sometimes becomes slightly swollen. The plastids are scarcely altered, if one leaves out of account their reduction in size concurrent with the ageing of the leaves; they are often filled with starch, occasionally with more than is present in healthy cells. Thus, the host cells normally remain alive; sometimes 12 days after infection not a single cell has died and, in old infections (more than 16 days old, i.e. after production of the new uredosori), only 1-2% of the host cells, at most, are dead. Parasite and host present an appearance of marked compatibility. Mutual disturbance and injury are at a minimum. The parasite lives freely and the host suffers

little disadvantage in spite of its task of sheltering and nourishing the parasite; a balance is established between the two, they tolerate each other and the outbreak of disease is benign (*schön*).

The situation is different in the hyper-susceptible variety Malakoff of *Triticum vulgare erythrospermum*. Here, the communal life is parabiotic. The first stages of the infection resemble those in the highly susceptible variety Little Club, except that the guard cells in contact with the fungal hyphae suffer greater injury and often die. When, however, the hyphae are no longer able to live on their own resources and send haustoria into the mesophyll cells, the latter often react with the utmost intolerance, they degenerate and die with symptoms of gummosis. Fig. 199 illustrates this phase in another rust disease (black rust).

This intolerance (incompatibility), from a parasitological point of view, affects both partners disadvantageously. With regard to the parasite, its haustoria remain small and, at most, reach a length of 16μ and its nucleus and protoplasm disintegrate in 1–2 days to form a practically homogeneous, darkly coloured mass (Fig. 197, 5, *H*); the majority of the invading rust mycelia, even at this early stage, therefore, perish from exhaustion (Fig. 197, 6), they starve. Others manage to survive and to attack a few new cells; in these the reaction is less vigorous than at the start of infection, so that the host cells and the haustoria remain together for at least a few days and nourish the parasite sparingly. The hyphae are, however, poor in content, the older portions become empty and often inflated and misshapen, and only the terminal segments remain alive, but their protoplasm is vacuolate even at the tip; they are quite obviously struggling to exist. Under these unfavourable conditions of life the mycelia reach a diameter of only about $100\text{--}300\mu$, and only in especially favourable cases are they able to produce a few uredospores.

In the local area affected this incompatibility is equally disadvantageous to the host; in extreme cases the infected cells die at once, as indicated above. Yet the region of activity of the parasite is, at first, very circumscribed. The cells immediately bordering the site of infection are but slightly affected during the first 8 days; only later, when the incubation

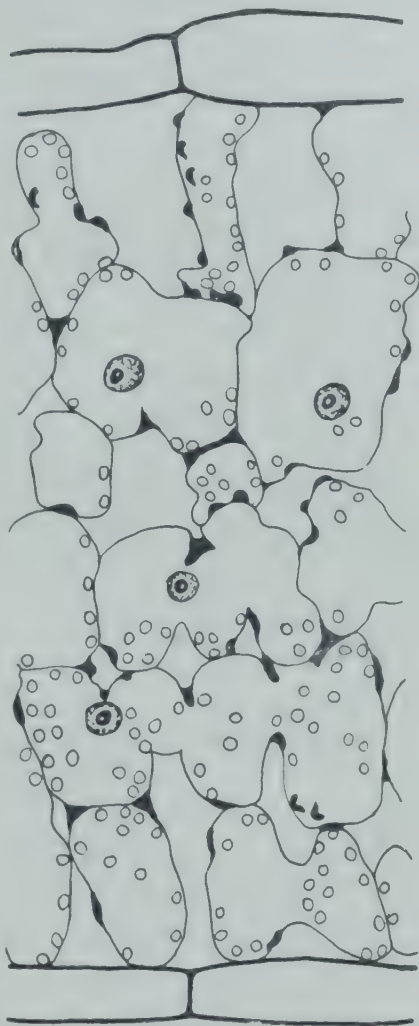


FIG. 198. The reaction of a variety of wheat hyper-sensitive to brown rust infection. Explanation in text. The portion figured is about 150μ distant from an 11-day-old locus of infection. Approx. $\times 365$. (After Allen, 1927.)

period is drawing to an end, do the pectin layers of the cell walls beneath, and adjacent to, the focus of infection begin to swell in a lobed or wart-like manner (Fig. 198). Surprisingly, the distant action of the parasite, therefore, in these cases only becomes visible in the cell walls, not in the protoplasts which, cytologically, show no change.

Injurious as this over-sensitivity is for the hyperergically reacting cells, it is yet very favourable to the plant as a whole since the focus of disease is localized and the parasite is, to all intents, prevented from reproducing itself. The hyper-sensitivity arrests or prolongs the transition from infection to generalization. The fungus is able to take hold but is rendered harmless before it can develop its pathogenic capacities.

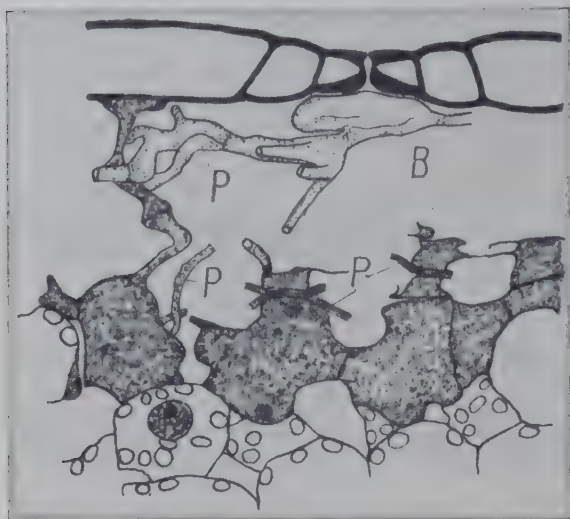


FIG. 199. The hyperergic reaction of Kanred wheat to an infection by *Puccinia graminis tritici*, biotype I. B sub-stomatal infection swelling. P fungal hyphae, partially aborted. Approx. $\times 350$. (After Allen, 1923; from Gäumann, 1944.)

In principle, the data are similar to those for *Synchytrium*. Both varieties of wheat are susceptible to infection by the pathogen and, in both, the plasmatic defence reactions are inadequate to destroy the fungus during the process of penetration; hence, the infection 'takes'. Further, in both varieties, the plasmatic defence reactions continue after infection, weaken and injure the parasite, and, in any case, ensure that it cannot spread unhindered through the host tissues.

As in the *Synchytrium* example, so in the case of rusts the two varieties of wheat differ only in their secondary behaviour. The compatible, normally susceptible, and only normergically reacting variety tolerates the parasite, shelters it, allows it to reproduce itself, and, therefore, becomes diseased; hence, it is disease susceptible. The incompatible, hyper-sensitive variety does not tolerate the parasite, it reacts idiosyncratically to its excretions, responds hyperergically, and thereby hinders or stops generalization of the parasite; in consequence, the plant develops only small local foci of disease. Practically speaking it is, therefore, disease immune. Thus, in these two examples, almost the same causes lead to diametrically opposite effects.

In contrast to the *Synchytrium* example, the hyperergic reaction of the tissues in the neighbourhood of the site of rust infection is not complete and uniform. Hence, a closed necrotic area as in Fig. 190 does not usually occur round the focal point, but the sensitivity of the different cells clearly exhibits individual variations so that, within this unstable zone of conflict, scattered cells respond hyperergically. The focus of infection is, therefore, seldom completely isolated, but at most retarded in its spread.

These individual differences in the sensitivity of single cells are more obvious in the case of black rust (Table XLVII) in which the various cell groups react differentially even to different strains of the same parasite. The normergic plasmatic reactions and the hyperergic necrogenous reactions of individual cells or cell groups are here so closely interwoven that it is not possible to determine how much of the final result is due to the former and how much to the latter. Together with the structural resistance factors they constitute a whole or a totality, viz. the capacity for resistance of the given wheat varieties against the given strains of parasite under the given conditions.

TABLE XLVII

Summary of parasite host relationships of biotypes 9, 21, and 27 of Puccinia graminis tritici, in Triticum dicoccum (Khapli emmer). (After Allen, 1926)

<i>Characters</i>	<i>Biotype 9</i>	<i>Biotype 21</i>	<i>Biotype 27</i>
No. of infection tubes which have penetrated . . .	52%	42%	21%
Injury to stomata . . .	very slight	medium	very severe
guard cells normal . . .	90%	53%	23%
guard cells slightly injured .	10%	19%	18%
guard cells killed . . .	0	28%	59%
Neighbouring epidermal cells affected	0	0	12%
Attainment of equilibrium between parasite and host .	average	slow	rapid
Alteration in haustorial mother cell	very marked	slight	absent
Incubation period	4-5 days	4 days	5-6 days
Final diameter of infection focus	2-3 mm.	2 mm.	3-7 mm.
Behaviour of host cells beyond mycelial zone	impoverished	collapsed	impoverished
Reproductive capacity of the parasite	± minute uredosori	uredosori more numerous	sterile (no sori)

In retrospect, it is clear that successful active defence against parasites also occurs in plants under certain conditions, viz. against the relatively numerous parabiatic parasites. Hence, infectious diseases do not play a larger role in the realm of plant life than in the realm of human life.

The difference lies only in the path by which this goal is reached. In plants, the normergic plasmatic defence reactions are generally rather ineffective and able only in favourable circumstances to localize, let alone eliminate the parasite, but if parabiatic organisms be involved the pathological hyperergic reactions step into the breach, compensate for the inadequacy of the normergic defence reactions, and cause the infection to abort together with the affected tissues. Man, on account of his adequate normergic defence reactions, does not need spontaneous hyperergic reactions which, therefore, play only a slight part in the study of human immunity.

Corresponding to their primary significance in the occurrence of plant disease, the necrogenous defence reactions form a valuable basis for breeding infection-free, economic plants (breeding of disease-immune, rather than of disease-resistant varieties, p. 297). Since breeding for true immunity (left-hand limb of curve in Fig. 193) offers only a distant prospect of success, in many cases plant breeders of to-day go to the other extreme and try, on the basis of the narrow right-hand limb of the curve in Fig. 193, to produce apparently immune varieties, which remain infection free because they react hypersensitively to the disease agent. This goal, as indicated by Roemer *et al.* (1938), is attempted to-day in the improvement of numerous agricultural and horticultural plants. Examples are the breeding of vines resistant to *Plasmopara viticola* (downy mildew: spot infection due to aborting reactions, Husfeld, 1933; here, too, the mycelium shows no symptoms of starvation, Lepik, 1931); the breeding of apple trees resistant to *Endostigme inaequalis* (apple scab) (M. Schmidt, 1938); and in breeding barley against *Erysiphe graminis hordei* (powdery mildew) (Honecker, 1934). In barley, the disease resistance of the variety is proportionate to the speed with which the attacked epidermal cells become necrotic (see Fig. 194); their early death deprives the pathogen, which is an obligate parasite, of its source of nourishment and so renders it unable to spread in the tissues and reproduce itself.

(b) *Induced Anti-infectional Defence Reactions*

In the autonomous anti-infectional defence reactions so far discussed, both the reactivity and the immunological efficiency are wholly natural to the host; they are innate specific characters. Among the manifold reactions which the pathogen evokes in attacked individuals and which are usually harmful to them, there are occasionally a few which have an opposite effect. Instead of weakening the host they stimulate it and awaken or activate in it a readiness for defence previously only dormant or present in inadequate degree (p. 280), with the result that it can now do things of which it was formerly incapable. The newly appearing defence reactions are called induced anti-infectional defence reactions.

The disease, which will now be called the pre-disease, thus conditions in the affected individuals a change, a sensitization, a heightened reactivity of certain tissues or of the whole organism. This new complex has an allergic character and is one of the few examples of allergy known in botany.

So far as normergic defence reactions are concerned in this reinforcement, they appear to retain their normergic character; a change-over to hyperergic activity (induced anaphylactic shock) has not yet been established with certainty.

In human medicine, sensitization leading to protection against re-infection is a reaction either to the pathogen itself or to its metabolic products.

Sensitization by the parasite itself occurs spontaneously in man through

the survival of diseases of childhood. Most of us, as children, sicken with measles, German measles, diphtheria, or whooping-cough, and recover because of our spontaneous defence reactions; we are, therefore, sensitized against them and protected from re-infection in later life. This process (sensitization or active immunization by the pathogen) is imitated clinically, e.g. in small-pox inoculation: here the small-pox virus, altered by passage through an animal body, is inoculated into the human body where it liberates an excess of protective substances ('reserve defensive substances') and, through sensitization, also engenders a heightened disposition to defence, so that later the body is better able to withstand a fully virulent infection.

In medicine, prophylactic tetanus and diphtheria inoculation are based on the second possibility, sensitization or active immunization by means of the metabolic products of the pathogen. In this, a toxin or one poisoned with formaldehyde (antitoxin) is injected into the body and stimulates it to specific anti-infectious defence reactions.

Both types of induced defence reaction, (*aa*), sensitization by means of the pathogen, and (*bb*), sensitization by means of its metabolic products, are also known in the plant kingdom.

(*aa*) *Sensitization of the Host by the Pathogen Itself*

In this first type of induced immunity, the pre-conditions in the plant kingdom are very different from those in human and veterinary medicine. In the two medical examples just noted, spontaneous diseases of children and small-pox inoculation, it is the process of recovery from infection, auto-sterilization, which engenders the altered reaction norm of the organism. The pathogenic agent disappears from the body which, however, retains a heightened disposition to resistance against subsequent re-infection.

This form of acquired immunity can hardly be expected to occur among plants because the plant body cannot, as a rule, free itself entirely from infection. In principle, infectious diseases of plants are incurable. Plants can, however, achieve another kind of induced immunity, also first recognized in human medicine although here it is only of minor importance, viz. immunity associated with infection or 'premunity' (French, *prémunition*, generally known as infection immunity). In this phenomenon an organism, or certain of its tissues, cannot be successfully super-infected by a second infection as long as the first continues to exist. In acquired immunity, recovery from infection protects against re-infection whereas, in premunity, an existing infection protects against super-infection.

Premunity, therefore, like acquired immunity, is based on an alteration in the host due to pre-infection. In consequence of this, the plant becomes endowed with capacities it previously did not possess but, in contrast to acquired immunity, the modification of the host in premunity lasts only as long as the pre-infection continues.

According to their topological fields, local and humoral premunity

may be distinguished. In local premunity, only the tissues directly invaded and their nearest neighbours are protected against super-infection, whereas in humoral premunity (p. 315), the local infection protects the whole organism.

(a) Local premunity (French, *préséance*; German, *Vorrang*). Local premunity depends on the fact that the primary infection modifies and sensitizes the substrate, the host tissues in its neighbourhood, and, in consequence, this region or its immediate vicinity is barred to a later second infection. It is not, therefore, the original pathogen itself which obstructs subsequent competitors, but rather the host plant which is



FIG. 200. Vaccination figures of *Phytophthora infestans* in potato tubers. Explanation in text. Approx. $\times \frac{2}{3}$. (After Müller and Börger, 1941; from Gäumann, 1944.)

stimulated by the first infection to defend its priority rights. This induced protection holds only for the specific pathogen or for related pathogens of the same species group.

The conception of *préséance* has been developed particularly by dermatologists. Thus, in the secondary stage of syphilis, a mild fresh infection does not 'take' since the body is already protected by the existing general infection. Only a severe super-infection can overcome the resistance but, even so, it is unable to evoke the primary symptoms. The body has been altered by the existing general infection and no longer gives the normal reactions to primary infection. This protection and this modification endure only so long as the original infection exists and disappear with its cure.

According to its field of action, local premunity in plants can engender either an immunity from infection (especially in fungal diseases, mycoses), or an immunity from disease (especially in virus diseases, viroses).

A classical example of local immunity from infection is afforded by Bernard's experiments (1909) with orchid mycorrhiza. If young orchid embryos (Fig. 182) are infected by a mildly aggressive strain of mycorrhiza, the hyphae penetrate the place of attachment of the suspensor to a depth

of a few cell layers and are then checked. If, a few days later, an aggressive strain, so virulent that it would normally kill the seedlings, is brought into contact with the infected embryos, it is no longer able to make use of the entry point and infection does not 'take'. However, when the seedlings have developed hairs (bristles; Fig. 182, right), the fungus can penetrate these and initiate a lethal infection some distance from the original focus of disease. The capacity of the embryos to resist has, therefore, been locally sensitized by the mild pre-infection. This local premunity is group-specific; if, for instance, it be initiated by a weak strain of *Rhizoctonia repens*, it is effective even against aggressive strains of *R. mucoroides* and *R. lanuginosa*.

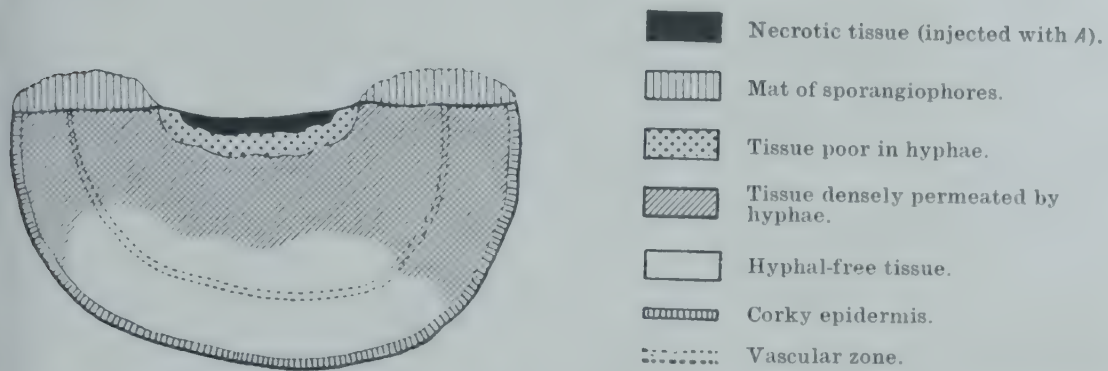


FIG. 201. Distribution of intercellular hyphae of *Phytophthora* in a pre-treated potato tuber. Explanation in text. (Diagrammatic after Müller and Börger, 1941.)

The vaccination experiments of Müller and Börger (1941) with the *Phytophthora* of potato late blight depend on the same principle. *Phytophthora* immunity of potatoes is based on the fact (p. 299) that, after the failure of the plasmatic immunity reactions in the affected tissues, extraordinarily rapid hyperergic necrogenous reactions set in, which destroy the parasite. In the experiment shown in Fig. 200, the cut surface of one half of a tuber was inoculated in the form of a cross and the other half in a horizontal band, with the parabiotic fungus strain *A*, against which tubers of type *F* (Fig. 194) are disease immune; 16 hours later the whole cut surface was sown with the sporangia of the eusymbiotic fungus strain *S*, which sets up only normergic reactions in the tuber and, therefore, infects it strongly. After 5 days (at 19 °C.) a vigorous white mycelium of strain *S* had developed on the untreated surface whereas, in the vaccinated areas, the super-infection had not 'taken'.

Even beneath the necrotic layer a portion of the tuber parenchyma remained devoid of hyphae for some distance (Fig. 201). It is difficult to imagine that necrotic substances had diffused down into these tissues in sufficient quantity to protect them against the fungus, since these substances are, for the most part, excluded by the semi-permeability of the protoplasm. Evidently, the few metabolic products of the parasite and the necrogenous substances of the aborting tissues, originating in the local disease focus and allowed through the protoplasmic membranes, have sensitized the parenchymatous cells in question and induced in them

a heightened defence readiness, so that they are now able to inhibit the development of the parasite.

Furthermore, Beauverie (1901), Ray (1901), Carbone and Kaliaew (1932), and others (Baldacci, 1942, p. 43 et seq.), treated begonias, &c., with enfeebled or weakly pathogenic strains of *Corticium vagum* var. *ambiguum* (Baldacci and Cabrini, 1939; generally mistakenly referred to as *Botrytis cinerea*), and, thereby, obtained in the affected plant an increased power of resistance against highly virulent strains. However, the extent of the zone of induced protection is not known with certainty; does the



FIG. 202. Mottling in a leaf of the potato, var. Prussian, due to X virus. Approx. $\times \frac{3}{2}$. (After Köhler, 1940; from Gäumann, 1944.)

local infection release a humoral sensitization, or is the effect limited to the immediate neighbourhood of the tissues previously diseased?

In the examples so far discussed (exclusively mycoses), premunization (so far as is known to-day) acts directly against super-infection. Thus the affected plants have become locally resistant to infection as a result of previous disease; they possess an acquired immunity against infection.

The circumstances affecting premunity in the viroses are different. The picture differs from that in the mycoses because, in the viroses to be discussed, the infective material floods the whole body, everywhere

releasing local, cellular defence reactions and thereby simulating humoral immunization. That it is, however, only a cellular or tissue effect is shown by the occurrence of super-infections. The primary, protection-inducing virus is not always distributed equally throughout the plant body, hence, the induced protection is not complete everywhere so that, occasionally, at one place or another, super-infection may develop to a limited extent.

A specific, local immunity to disease (i.e. one in which the still existing primary disease is effective only against a fresh infection by another strain of the same virus) is exemplified, for instance, by aucuba mosaic and by the potato virus X group.

If young tobacco plants with aucuba mosaic (disease illustrated in Fig. 203, right), are kept for 3 days at 35° C. the virus mutates (sometimes clearly under the influence of the host) and weakened strains arise, which are characterized, for instance, by the production of less intensive mottling of the leaves or, on occasion, by a loss of capacity to initiate necrotic processes. If other tobacco plants be inoculated with this weakened mosaic virus, they will not be affected later by the fully virulent virus (Kunkel, 1934);

the latter is unable to multiply in the tissues, or does so only to an insignificant extent, and in consequence the plants no longer become diseased.

Potato virus X is a group of viruses characterized, among other properties, by being of similar dimensions and having a similar thermal lethal zone (between 68° and 75° C.), by being directly transmissible to other plants by means of the sap of the host (rubbing, pricking, or grafting) and not indirectly by means of the peach aphid (*Mysodes persicae*), and by dis-



FIG. 203. Premunization of tobacco. Left: tobacco leaf pre-infected with common tobacco mosaic. Right: healthy tobacco leaf. Both leaves subsequently infected with aucuba mosaic. The disease breaks out only in the healthy leaf. Approx. $\times \frac{1}{2}$. (After Kunkel, 1934.)

organizing the chlorophyll in the interveinal spaces of the leaves (Fig. 202) or, in severe cases, by producing local necroses.

In the premunizing experiments of Salaman (1933, 1938) about to be discussed, a weak *G*-strain was used as the protective virus; this frequently produces no symptoms (apathogenic) but may give rise to a pale, scarcely visible mottling of the leaf tips of suitable test plants. In one case, an *L*-strain was used as the pathogenic virus for super-infection; this is a moderately strong virus which induces pale yellow mottling and dwarfing. In a second case the *S*-strain was used; this is a vigorous strain of the classical potato mosaic virus which produces light green coloration of the interveinal spaces, so that the leaf veins lie embedded in dark green bands.

The tests were made with tobacco plants since these are better able to withstand greenhouse conditions than are potatoes. The layout of the experiment was as follows. A number of tobacco plants was pre-infected with the weakest *G*-strain to serve as a protective virus; the symptoms are so slight that there is only a suspicion of mottling. At intervals of 24 hours successive pairs of groups of these infected plants were super-infected with a vigorous *L*- or *S*-strain serving as the pathogenic virus. In the

super-infections made during the first 4 days, the *L*- or *S*-strains penetrated and the plants became diseased according to the *L*- or *S*-strain pattern. On the other hand, those plants super-infected on the fifth day evinced a partial protection, the pale yellow mottling of the *L*-virus being limited to small isolated portions of the leaf. Super-infection carried out on the eighth day no longer produced either *L*- or *S*-disease and the plants reacted according to the *G*-type only. The *L*- or *S*-virus could no longer develop in them and remained limited to the site of infection: these plants, therefore, were protected against disease from these two viruses.

The effects are even more striking in the case of *Datura Stramonium* (thorn-apple). If this receives a dose of *G*-virus, it is protected against the *L*-virus and another necrotic form of X-virus, both otherwise lethal; again, the viruses are unable to spread within the plant.

These facts suggest the following considerations.

1. The significance of induced protection. Pre-diseased individuals remain susceptible to virus super-infection, in contrast to the mycoses just discussed. The super-infection therefore 'takes' but cannot spread. As a result of sensitization, the host is enabled to prevent the transition from infection to generalization (previously it was unable to do so): it localizes the infection and it no longer possesses any proneness to disease and, therefore, in spite of its susceptibility to infection, it has become resistant to disease (acquired disease immunity).

This situation is similar to that obtaining in medicine. In diphtheria, for example, human beings remain equally susceptible to infection after surviving an earlier attack but, on account of a changed reactivity, no longer become ill; disease immunity has been acquired in consequence of the pre-disease. But the subsequent fate of the second infection is different in the cases of diphtheria and plant viroses. In diphtheria, the second infection, on account of our great recuperative power, is, in course of time, again eliminated, whereas, in plant viroses, the super-infection, on account of the lower capacity of plants for recovery, cannot be eliminated and the super-infecting virus continues to remain infective at the site of infection; it is neither destroyed nor inactivated by the host. If, for example, the affected tissues are cut out and laid upon plants not pre-diseased, the latter will develop the typical symptoms; only in a previously diseased host is the super-infecting virus unable to multiply and spread. Thus, it is not the ability to recover but the power of localization which is augmented in the test plants by pre-disease.

2. The specificity of induced protection. The protection afforded by the *G*-strain is specifically limited to the X-group of viruses and is powerless against either potato virus Y or the common tobacco mosaic, or against the virus of ring spot necrosis (Fig. 223).

In other viruses, local premunization acts group-specifically: the still existing pre-disease due to a given virus is protective not only against a fresh disease by another strain of the same virus but also against diseases induced by related viruses (Thung, 1932; Kunkel, 1934; Caldwell, 1935; &c.).

If mature and healthy tobacco leaves are inoculated with aucuba mosaic, they show necrotic mottling as the primary effect (Fig. 203, right); if however, they have been pre-diseased with common tobacco mosaic (Fig. 203, left) or inoculated at least 2 days previously with this virus, then the aucuba virus will be limited to the point of infection; it 'takes' and is neither destroyed nor eliminated by the defence reactions of the host although it can no longer multiply or spread, or do so only insignificantly.

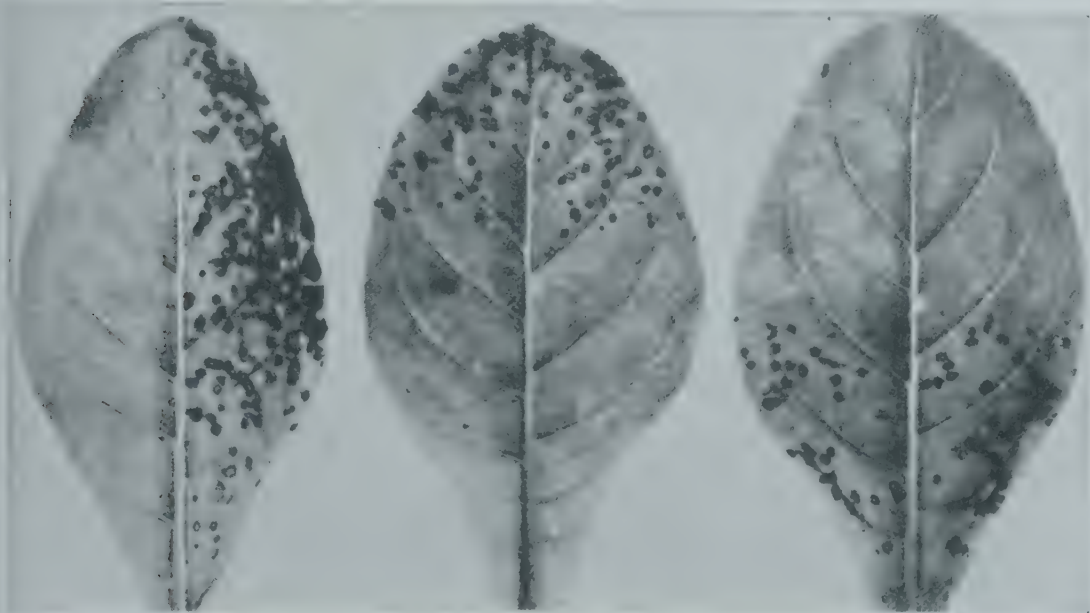


FIG. 204. Localization of the protective effect in pre-diseased tissues. Explanation in text. Approx. $\times \frac{1}{2}$. (After Kunkel, 1934.)

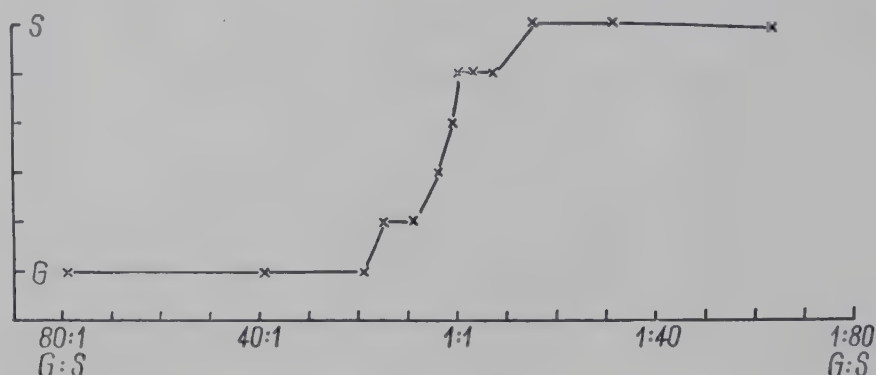
Premunization is essentially limited to the infected zone. Fig. 204 depicts leaves, certain portions of which were rubbed with common tobacco mosaic virus and 5 days later the whole leaf surface was treated with aucuba virus; aucuba appeared only in the portions not previously inoculated, yet it is not merely the directly infected cells that are protected, but also non-infected neighbouring cells for a distance of about seven rows. Premunization is, therefore, not strictly limited to the previously diseased cells but affects (indicative of humoral premunization, to be discussed later) non-infected neighbouring cells and this produces the beginning of an infectional immunity of the tissues.

Furthermore, the protective effect is group-specific to aucuba mosaic but not, for example, against a special necrogenous form of common tobacco mosaic. By using such crosswise premunization tests it is possible to determine the genetic relationships between viruses; to some extent these are a substitute for the serological determination of relationships in research on animals. We shall return to this question when dealing with induced tolerance.

In other cases there is the possibility of a cumulative sensitization. Tobacco plants, pre-infected either with 'severe mosaic' plus ring spot

necrosis (Fig. 223), or with ring spot necrosis plus 'distorting' mosaic, are no longer susceptible to 'white mosaic' disease, whereas none of these three viruses alone protects against 'white mosaic' (Thung, 1936).

In all the instances of premunization so far known, there must be a minimum time interval between the protective primary infection and the pathogenic super-infection that is to be warded off. Thus a certain latent period is necessary for sensitization of the pre-diseased tissues; about 16 hours for potato late blight (p. 309), 2 days for common tobacco mosaic virus, and about 8 days for potato virus X. If this period is reduced, primary infection affords little or no protection against super-infection.



In the quantitative relation 79 *G* : 1 *S* to 19 *G* : 1 *S*, the *G*-virus was able, through the sensitization of the host, to prevent the development of the *S*-virus simultaneously introduced and, conversely, the *S*-virus proportions of 1 *G* : 15 *S* to 1 *G* : 63 *S* blocked the development of the simultaneously inoculated *G*-virus. In the mixtures between these values both viruses developed simultaneously in the host plant.

The explanation of these remarkable relationships is not consistent with the theory that the viruses inactivate each other since they are fully infectious if they are separated mechanically or by means of differential hosts. Blocking depends much more on the sensitizing of the defence readiness of the host plant by the quantitatively stronger partner and on its quantitative prepotency masking the absence of the latent period, and on the hindering of the further development of the quantitatively weaker partner by increased production of antibodies by the host.

(b) Humoral premunity. In humoral premunization the existence of a local infection protects the whole organism against disease produced by the same or a related pathogen. The induced protection is effective only so long as the primary infection continues.

Koch's phenomenon in tuberculosis provides a classical example in human medicine. A body in which a local primary tubercular lesion exists becomes sensitized by it. It is not, however, protected against a super-infection with tubercle bacilli; thus, if a second tubercle infection occurs in some other part of the body, this infection will 'take', but the body now reacts differently. Instead of a lesion which persists and gives rise to secondary lesions, an allergic reaction occurs at the new focus which eliminates the second infection. Hence, the transition from infection to generalization is prevented as a result of the existing pre-disease; the existing local pre-infection protects the whole body against disease arising from super-infection.

The botanical investigation of this type of premunity is still incomplete. That sensitization is not strictly limited to the infected cells but may extend for a distance of about seven cells in the non-infected neighbouring tissue has already been demonstrated in the premunizing group of tobacco mosaic - aucuba mosaic (p. 313). The question, therefore, is primarily one of the topological extent of the sensitization effect.

The pear mistletoe appears to be an example in which a still-existing, strictly localized disease gives rise to a general humoral sensitization. Pear trees, in contrast to apple trees, only rarely harbour mistletoe plants (*Viscum album*), and certain varieties never do so. This is due to special defence reactions of the tree (Heinricher, 1917, 1929). If susceptible varieties of pear, e.g. Good Louise of Avranches and Good Grey, are invaded by seedlings of the mistletoe of deciduous trees, or if fresh mucilage from the berries is smeared on them, they react hypersensitively with gum, cork, and bark formation. In consequence of this, the unhealthy tissues of the branch are insulated from the healthy tissues so that the parasite is cut off from the sap of the host, withers, and falls off together with pieces

of bark from the host (Fig. 206). In some cases the branch dies completely; hence, on these pear varieties, the occurrence of mistletoe is exceptional.

In these pear varieties, therefore, the malignity of the mistletoe for its host is as great as that of *Synchytrium* (p. 291) and, as in potato wart disease, the defence reaction of pear trees also consists of a mixture of eliminative and necrogenous defence reactions.

What happens if the healthy branches of a pear tree of this reaction type on which mistletoe very seldom grows are again sown with mistletoe seeds (super-infection)? The histogenous eliminative and necrogenous reactions are only weak but no infection takes place, evidently because of a heightened readiness for cellular defence: the whole tree has been sensitized in its defence readiness by the infected branch. Because of the great technical difficulties it is not easy to carry out experiments but it would be interesting to know how long the protection against mistletoe lasts, once the mistletoe-bearing branch is cut off.



FIG. 206. Scab-canker reaction of a variety of pear susceptible to mistletoe infections. Nat. size. (After Heinricher, 1917.)

In the remaining examples of humoral premunity in plants, as is also true of the bacterioses (Savulescu, 1936, &c.) the positive and negative data roughly balance. Bacterial crown galls caused by *Bacterium tumefaciens* (Fig. 67) will next be considered. E. F. Smith *et al.* (1911) infected *Chrysanthemum frutescens* with a strain of this bacterium, took cuttings from the diseased stock which, in turn, were infected, again took cuttings to be infected and so on, and thus produced an increasing immunization of the clone. Even the third generation of cuttings showed increased immunization recognizable by the very slow growth and small size of the tumours, whereas the same bacterial culture, simultaneously inoculated into fresh (not previously diseased) chrysanthemums, produced the large characteristic galls in a short time. Similarly, Arnaudi (1925) observed that in pelargoniums no super-infection 'took' within some centimetres of an already-existing tumour, and (1928) that fresh stocks generally showed a greater susceptibility than previously diseased ones. Duyfjes (1935) noted that even the youngest tumours on pelargonium and *Bryophyllum crenatum* inhibited the development of fresh infections (super-infections); this cannot be the result of a simple inhibition of vegetative growth due, for example, to competition for nutriment. Magrou (1935) found that new tumours did not develop easily on super-infection of pelargoniums and that the plants occasionally showed hypersensitive manifestations.

On the other hand, N. A. Brown's experiments (1923) were unsuccessful even though in two cases a transitory immunity was obtained: she attempted to build up a lasting active immunity in chrysanthemums and roses by

alternate gall infection and subsequent vegetative propagation. Riker (1926) could not detect any difference between the reaction type of healthy and pre-diseased clones or find any agglutinins, precipitins, or lysins in the tumours. Manil (1936) found no diminution of infection, i.e. no protection against disease, when tobacco was super-infected with *Bacterium tumefaciens* (or with *Bacterium tabacum*) after pre-inoculation of the leaves just below with the same bacterium. Nevertheless, we should not simply disregard the positive observations. Evidently, a general or local modification of the host which renders it able to resist a new gall infection is only possible under special conditions (variety, age of stock, nutritional state, &c.).

The observations on another bacteriosis, the root nodules of Leguminosae, are equally contradictory. If plants of white clover (*Trifolium pratense*) and pea (*Pisum sativum*), which already carry nodules of a given strain of *Bacterium radicicola* on their older roots, thereafter form new roots, the latter withstand super-infection with a second strain much better than do completely healthy plants (Dunham and Baldwin, 1931). The nitrogen fixation and yield of leguminous plants spontaneously pre-infected with an inefficient bacterial strain are, therefore, only very little increased when the plants are later super-infected with a more efficient bacterial strain. On the other hand, contamination of a good crop by a bacterial strain of little value does not lead to a corresponding decrease in production. However, in the case investigated by Löhnis (1930), again *Trifolium pratense*, the decreased liability to infection of the plants bearing root nodules can only be ascribed to the nitrogen which is made available to the host by the bacteria and which, like artificially added nitrogen (p. 282), reduces the susceptibility caused by partial undernourishment of the plants. If roots are pre-infected with a bacterial strain which fixes little nitrogen, this produces no immunity against strains which are good nitrogen fixers.

The fact that, in certain tree diseases, epidemics often disappear in the course of time can be used as a further argument in favour of premunization of the whole plant by local infections. For example, when the oak mildew (*Microsphaera alphitoides*) spread pandemically in 1907 and 1908 from France over Europe and western Asia, the injury to young oaks reached considerable proportions. In the following decades the severity of the disease diminished, perhaps because, as in mistletoe immunity, protective substances passed from the leaves into the trunks, and in the following years the leaves became more resistant. Montemartini (1930) inoculated six young oaks with oak mildew and left six others healthy; in the following year he inoculated all twelve, with the result that the second six became diseased while the first six, with one exception, remained healthy.

We have repeated these trials with *Quercus Robur* without, however, observing in equally vigorous individuals a decrease in susceptibility. This, again, shows the difficulties of making investigations; the experi-

ments are valid only for the given conditions, which are often hard to define and, therefore, far from easy to repeat. There is no doubt that the disease produces an internal modification of the inoculated trees; thus Orsenico (1939) obtained a conidial germination of only 10–20% in expressed sap of oak leaves which were already severely infected, whereas spores in sap from healthy trees gave a 50% germination. This proves only the existence of inhibiting substances; it does not prove that these will be effective in the tree in the following year.

In coffee bushes, also, the later waves of infection by *Hemileia vastatrix* (coffee rust) are considerably lighter than the first wave, so that the number of falling leaves and the number of rust sori on the same bush clearly diminish (Dowson, 1921). The bushes thus appear to develop a certain immunity as a result of repeated infection: the parasite retains its aggressiveness and appears with its original intensity in neighbouring plantations when these are attacked for the first time. However, this decrease in infection can also be explained on the basis of a diminution in the vitality of the coffee bushes. As will be seen later, a lessened vitality of the host conditions a reduced disposition to rust infection, and conversely. When, therefore, during the first infection wave, the bushes lose all their foliage each year and are induced to form accessory branches, the weakening thus brought about might explain the decrease in rust infection without any need to hypothecate special defence substances.

Apart from the case of mistletoe, humoral premunization of plants through local infections has, therefore, not yet been conclusively proved, in contrast to human medicine. It appears as if it were difficult for plants to react as a whole against local infections. The 'objective', factual, and measurable phenomena of premunity in plants, like the autonomous defence reactions, seem to be, in the main, only local in character.

(bb) *Sensitization of the Host by the Metabolic Products of the Pathogen*

Unlike sensitization by the pathogen itself, this second type of sensitization cannot occur naturally in the field but depends exclusively on clinical or laboratory methods; in positive cases it will serve as a valuable contribution to the problem of anti-infectional defence reactions.

This mode of sensitizing plants is, however, technically very restricted. Since plants lack a closed circulatory system, metabolic products of the parasite thought to be antigens can only with great difficulty be introduced and distributed in the plant to be sensitized. If the infection occurs in exactly the same spot as the previous vaccination, it will be interfered with by the tissue reaction to the vaccination; if it occurs a few centimetres away, there is the objection that sensitization has not been able to reach so far, at least not in the given time.

The fundamental question is, therefore, whether plants are at all able to react in a more or less specific manner to introduced foreign substances. If standard tests with substances other than those formed by plant parasites are considered, the author has not been able to find, among the

numerous positive results in the literature, one which demonstrates a specific anti-reaction made under conditions that are precisely defined, above criticism, and repeatable. To pick out only a few examples: Lumière (1921) and Lumière and Couturier (1921) believed they had sensitized *Rumex* and hyacinth leaves and onion scales with horse serum, &c., so that on a second injection anaphylactic shock occurred; yet their findings were not confirmed by Nobécourt (1925), Otto and Herrig (1927), &c. Picado (1921) injected maize pollen into cladodes of *Opuntia* and believed he had observed new specific agglutinins and lysins in the expressed sap; yet Carbone (1925) obtained negative results on repeating these experiments. Tubercle bacilli inoculated into living tomatoes, &c., were, after two or more weeks, agglutinated *in vivo* and eventually disintegrated, whereas *in vitro* (in expressed sap) the corresponding reaction did not occur (Much and Nyrén, 1930; Much, 1931); owing to the complexity of the relations described by the two authors it is equally possible that pre-existing pseudo-antibodies were present, instead of acquired agglutinins. The same objection obtains in regard to the researches of Stickl (1927) who introduced typhoid bacilli into tulip leaves, later re-isolated them, and found them altered in form and in their serological reactions. Personally, I do not doubt that the plant body, like the animal body, is capable of more or less specific anti-reactions but, owing to the great technical difficulties, conclusive evidence is still lacking.

The auspices for experiments on the vaccination of plants with the metabolic products of their parasites are, therefore, not very favourable; however, or perhaps hence, numerous workers have been engaged on this problem and have used as antigens either dead cultures or cultural extracts or, in joint cultures on artificial media, the immediate excretions of the parasite itself.

For the first method, sensitization by dead cultures, experiments with *Bacterium tumefaciens* will be discussed. N. A. Brown (1923) killed cultures of this bacterium by maintaining them for 10 minutes at 60° C. and injected them into the vascular bundles of chrysanthemums and roses; these treated plants were later inoculated at the site of the first injection, or at the next higher internode, with the given bacterial strain. Whereas the controls developed small tumours after 14 days, the reaction in the internodes of the vaccinated plants took 3 weeks to develop; in direct infection on the site of injection, only two small tumours developed in nine plants (scarring reaction?). Thus there appears to be a certain disease-inhibiting effect produced by the vaccine.

Gheorghiu (1932, 1933) removed a piece of epidermis, 2-3 cm. wide, from geranium stems (*Pelargonium zonale*) and laid sterile cotton-wool, saturated with a bacterial culture killed at 60° C, on the cortical parenchyma for 12-18 days. A month later young shoots were inoculated without result with the corresponding bacterial strain, whereas normal tumours appeared on the controls. This induced protection apparently lasts a full 3 months. Nevertheless, Kaliaew (1935; cit. by Savulescu, 1936)

on repeating these experiments, observed no protective effect due to the vaccine, nor did Manil (1936) using a different technique.

The process of vaccinating plants with dead cultures of their parasites, therefore, does not yet seem to have led to definite success.

Again, in the second method, sensitization by culture extracts, the factual relations have never been demonstrated with certainty. Zoja (1925) and Leemann (1932) allowed wheat grains, &c., to germinate in a water extract of *Helminthosporium sativum* (leaf stripe) and later were unable to infect the experimental plants, in contrast to the controls, with the conidia of this fungus. Protection lasted from one to several weeks. The antigen is stable up to 50° C., but disintegrates on boiling. A similar sensitization for beans was effected by Nobécourt (1928) with extracts of *Botrytis cinerea* and *Bacillus carotovorus*; in the experiments of Kaliaew *et al.* (1935), an average of 42.2% of beans vaccinated with *Botrytis cinerea* remained alive, against only 4.3% of the non-vaccinated.

It might be objected that the slighter susceptibility of the vaccinated plants depends not upon an antigen-antibody reaction but rather upon a subsidiary effect such as saturation of the tissues with the metabolic products of the parasite, which by themselves exert a toxic effect (so-called 'staling products'). However, this explanation does not appear to meet the case because if such sensitized beans are killed, either with ether vapour or with carbon dioxide snow, *Botrytis cinerea* grows as well on them as on the control plants (Jarach, 1932); the increased resistance is thus bound up with the living cells.

On the other hand, Baldacci (1937) repeated Zoja's experiments and could not confirm the sensitizing action of the extract. In Manil's experiments (1936), beans which previously had been inoculated with the culture filtrate of *Bacterium syringae* (stem disease of lilac and numerous other trees and bushes) were found to be more susceptible to the bacterium than were the uninoculated controls. Hansen (1932) also failed to obtain any sensitization by inoculating seedlings of *Brassica Napus* with a culture filtrate of a strain of *Corticium vagum*. Only when using a non-sterile solution which had not been passed through a bacterial filter did he observe a slower appearance of the disease, but this was probably due to the antagonistic effect of other micro-organisms upon the parasite.

As in sensitization by dead cultures, there are both positive and negative findings, but the former indisputably carry weight. In future, the effectiveness of vaccination may, perhaps, be measured not only by the severity or mildness of infection, but especially by the more delicate events in the histological and cellular reaction of the host plants. Thus, Arata (1935) and Kaliaew *et al.* (1935) have observed a heightened vitality of the tissues of beans which had been treated with an extract of *Botrytis cinerea*; the histological defence reactions proceeded in the same manner but more rapidly than in the controls and, occasionally, hypersensitivity occurred.

For the third method, direct antigen activity by the living parasite, Bernard (1911) and Nobécourt (1928) have found an instructive example.

Loroglossum (*Himantoglossum*) *hircinum*, an orchid of the rough heathlands of the Jura, is infected in its roots instead of in its tubers, as is more normal in orchids, with a mycorrhizal fungus of the *Rhizoctonia repens* group. If this fungus be grown on agar medium, and an aseptic piece of sufficient size is cut from a tuber of the orchid and placed on the culture medium,

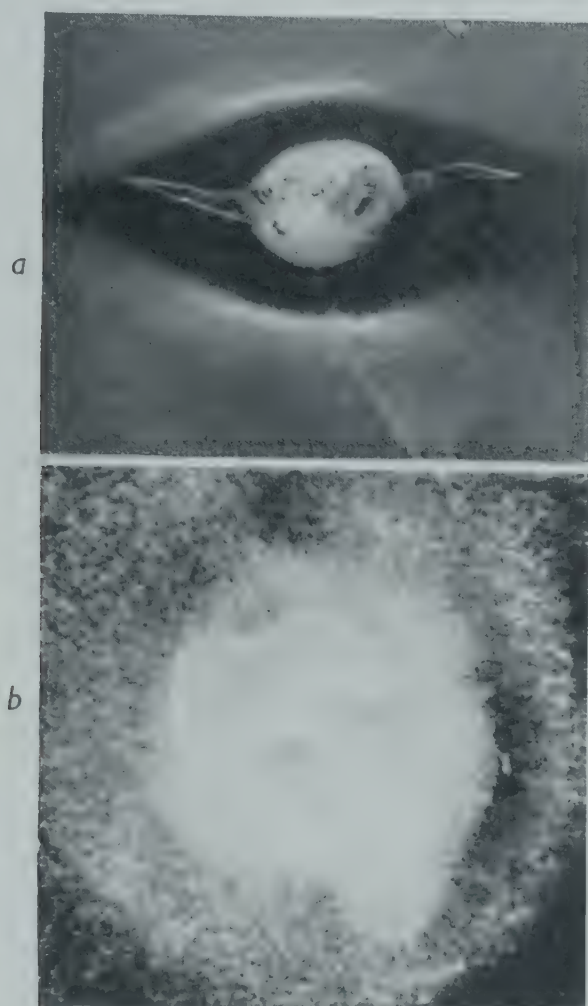


FIG. 207. Defence reactions evoked in the tubers of *Loroglossum hircinum* by the mycorrhizal fungus, *Rhizoctonia repens*. *a* a piece of living tuber; the fungus growing towards it from above and below is kept at a distance by defensive substances diffusing outwards. *b* a piece of dead tuber completely permeated by the fungus. Nat. size. (After Gäumann and Jaag, 1945.)

the growing fungus will be checked at some distance from the piece of tuber (Fig. 207 *a*), and, to a certain extent, growth will be arrested. Hence, some fungicidal substance must have diffused out of the cut piece of tuber into the medium. The question arises, is this substance originally present in the tuber or is it first produced as a reaction to the diffusing metabolic products of the parasite? If the tubers are previously killed, either by means of chloroform or low temperature (-10°C .; Fig. 207 *b*), the reaction does not occur; the fungus is not checked but grows vigorously through the tuber. The fungicidal substance is, therefore, actually a new formation by the living tissues, arising as the product of a true anti-

infectious defence reaction. Similar defensive effects can be obtained with *Orchis militaris* and *O. Morio* (Gäumann and Jaag, 1945).

The presence of these anti-infectious defence reactions is evidently the reason why the tubers are not, as a rule, infected by mycorrhizal fungi. However, these reactions can hardly be confined to the tubers; probably also occur in the root tissues to a lesser extent, so that they tend to limit the parasite to certain peripheral regions (p. 287) although they are not able to keep it completely at a distance as in the tubers.

(c) *The Mechanism of Anti-infectious Defence Reactions*

In the animal and human body the invading germs are combated on the one hand, by specialized defence cells which dissolve them enzymatically or incorporate and destroy them (phagocytosis) and, on the other hand, by biochemical means such as agglutinins, lysins, and similar defence substances in the blood serum. Phagocytosis is made possible by mobile 'devouring' cells, leuco-phagocytes, and by the stationary reticulo-endothelial system (e.g. certain supporting and vascular wall cells of the bone marrow, liver, and spleen). The mobile cells, particularly, enable the host to assemble hundreds of thousands of fighting cells at the threatened point in a short time and, by this high concentration, to eliminate the disease germs in this battle of cell against cell. Compared with this powerful protective army, biochemical anti-infectious defence measures play only a subsidiary part.

As a rule, such specialized defence cells are not found in plants; only the digestive cells of orchids (p. 288) are vaguely reminiscent of such a functional development. Mobile fighting cells, in particular, are inherently incompatible with plant structure on account of its rigid framework of cellulose walls. It is, therefore, not possible for a plant suddenly to concentrate the defensive potentiality of whole tissues or organs at the point of infection but every single infected cell or group of cells must carry out its own defence independently and without the support of mobile fighting cells drawn from distant tissues.

Hence, biochemical means of defence are all that are left to the plant, merely a fraction of the complex potentialities of our own bodies. In the human body, chemical defence plays only a subsidiary part of relatively slight importance, hence, it is not surprising that even less significant results are obtained in plants (mere localization, not cure of the infection).

The biochemical substances which are responsible for this defence are the same as those which were present in the cells before infection (p. 287) (pre-formed) but they then occurred in smaller quantity. During the invasion by the parasite the plant does not suddenly acquire a new potentiality which it did not possess earlier, it only reacts more vigorously than before; thus, the difference between post-infectious and pre-infectious specialized defence substances such as agglutinins, lysins, &c., is not qualitative but merely quantitative.

Not only are the functional means of anti-infectious defence much less

well developed in plants than in the animal and human body, but systematic research into the material basis of such defence measures in plants meets with far greater difficulties than in human and veterinary medicine. The medical man can obtain serum in unlimited amount and can follow the activity of his antibodies *in vitro*; he has them in his test-tubes. By contrast, botanists are in a comfortless position. Plants lack a central circulatory system with blood to transport the defensive materials; hence the botanist is unable to obtain reactive substances in quantities as great as those obtained from serum: painstakingly he attempts, in his experiments *in vitro*, to crush the cellulose walls of the given cells and tissues in order to express a small drop of juice and is delighted if this does not immediately become oxidized. Only in quite exceptional cases, therefore, does he obtain plant antibodies in the test-tube and, for the rest, he must draw his conclusions regarding their presence and activity indirectly from microscopic observations *in vivo*. Consequently the observations and experiments are difficult to repeat; hence, the many contradictions in the literature. It is but small consolation that even the medical man does not yet know everything about these problems.

Corresponding to these factual and technical difficulties our understanding of the anti-infectional defence reactions is at present very superficial. We understand neither the specific method of action of the antigens (whether fungi, bacteria, viruses, &c.), nor the mechanism of the production of anti-infectional defence products (which parts of the cells manufacture them, or how?), nor the mechanism of their chemical activity (any more than we know their chemical constitution). At present we understand only the more obvious ways in which they operate: according to these the active principles can be divided into four groups: (1) agglutinins, (2) lysins, (3) bacteriophages, and (4) virus antibodies.

aa) *Acquired Agglutinins as a Factor in Anti-infectional Defence*

Many plant pathogenic bacteria are able to induce the production of agglutinins in animal bodies, e.g. in rabbits or guinea-pigs (e.g. Goldsworthy, 1928, for *Pseudomonas cerasi* which causes a gum flux of sweet cherries in North America, and *Bacterium maculicola* the agent of a leaf spot disease of cauliflower; Carbone and Arnaudi, 1930, for *Bacterium tumefaciens*, &c.). Yet it has not been conclusively proved that they can also induce such reactions in plants, i.e. that flowering plants are able, like mammals, to respond to the presence of a foreign body, especially to the stimulus emanating from a parasite, by the production of defence agglutinins.

So far as *in vitro* methods are concerned, the practical difficulties in carrying out standardized experiments have been indicated on page 318. Even in experiments with actual diseased specimens, every positive statement can be matched by a negative one. Thus Schiff-Giorgini (1906) prepared sterile pieces of olive tissue taken from the vicinity of a tubercle and placed them in broth cultures of the pathogen *Bacillus Savastanoi*;

40 hours later agglutination of the bacteria had already begun, whereas controls, containing pieces of tissue from healthy trees, remained as tur as the original cultures. In this case, therefore, *Bacillus Savastanoi* appear to have immunized the substrate to some extent, in that the agglutination (assuming the absence of a masked bacteriophage) were adequate to precipitate some cubic centimetres of the bacterial suspension; Rist (1926), however, obtained negative results in a comparable investigation with the related *Bacterium tumefaciens*. Similarly, Cappelletti (1928) believed he had found newly formed specific agglutinins against *Bacterium radicola* in the root nodules of Leguminosae; they were thermolabile and were destroyed by heating for half an hour at 78–80° C.; but according to Israily et al. (1933; cit. by Savulescu, 1936) this was only a bacteriophage phenomenon.

In contrast to this failure of *in vitro* methods, microscopical observations *in vivo* (see pp. 283 et seq.) show quite clearly that some kind of agglutinative reaction must occur in the inoculated cells. If, for example, the parasite does not destroy its host cells either before or immediately after penetration but lives in them for several days or weeks, it frequently undergoes changes or deformations which can be explained only by the action of a newly formed agglutinin-like substance or by the increased secretion of already-existing agglutinins. Thus, Duyfjes (1935) established that similar degenerative phenomena take place in the bacteria of the gall caused by *Bacterium tumefaciens* as are found in the root nodules of Leguminosae (p. 284). Here, also, only the intracellular, not the intercellular, bacteria become deformed.

In fungal infections, deformation of the intracellular parasites, as mentioned on page 287 in the case of mycorrhizal fungi, is practically universal. So long as the hyphae invade the host tissues only intercellularly they grow normally but, as soon as lateral branches penetrate to the interior of the cells, they become changed into haustoria (Fig. 56) or coiled hyphae (Fig. 182), and growth ceases. The haustoria serve to take up nutrients and are, therefore, often assumed to be *ad hoc* organs or absorption cells. But this is incorrect; in artificial culture lateral hyphae also absorb nutrients and, in spite of this, do not become transformed into haustoria. Thus haustoria, as such, do not appear either in artificial culture or if the fungus continues to grow saprophytically in the tissues after the death of the host. Deformation arises only in consequence of the reaction of living host cells.

Moreover, the life of the haustoria is shorter than that of intercellular hyphae: they are successively corroded and digested. From the point of view of the fungus, they are not desirable organs but are products of deformation forced upon it because of the defence reactions of the host cells. These are artefacts produced during the struggle between host and parasite similar to the bacteroids in root nodules. Even with this explanation, the question is still open whether we are concerned with pre-formed substances in the host (normal or pseudo-agglutinins), or primarily with acquired agglutinins.

wly formed under the influence of the parasite; probably the latter is the case.

b) *Acquired Lysins as a Factor in Anti-infectional Defence*

Even though, as indicated earlier, the mechanism of animal or human phagocytosis cannot be expected to occur in the plant body on account of its different organization, yet flowering plants are able, in some cases, to dissolve the invading parasite enzymatically, a process functionally equivalent to phagocytosis.

The position is similar to that described for acquired agglutinins. It can readily be seen in microscopical observations *in vivo* that the lytic principle is active in the diseased tissues; e.g. in the digestion of the coiled hyphae of mycorrhiza (p. 287), of intracellular haustoria (p. 12), and of the bacteroids in root nodules (p. 285). Yet it is difficult to isolate the active principle *in vitro*, and, naturally, still more difficult to determine its chemical nature. Frémont (1933) thought he had found newly formed lysins against the bacteria when he vaccinated beans with *Bacillus proteus*, but Carbone and Alexandri (1935) could not confirm this finding.

Only an experiment by Burges (1939) can be cited as confirmed: he allowed the expressed sap from a tuber of *Orchis carnatus* to act upon the mycorrhizal fungus of this plant in a hanging drop, and observed both a toxic and a lytic effect (Fig. 208). But even here, it is not clear whether the substances in question were pre-formed or only produced after contact with the parasite (Fig. 207).

c) *Acquired Bacteriophages as a Factor in Anti-infectional Defence*

In bacteria sensitive to them, bacteriophages cause a loss of motility and hence a sedimentation in suspensions (agglutination), and further, during growth, an alteration in form and a dissolution (lysis). As already indicated, probably a number of the older observations on agglutinins and lysins as factors in resistance to plant disease actually refer to bacteriophages. However, on account of the great technical difficulties involved in their study the significance of bacteriophages in plant immunity is not clear even to-day.

There is no doubt that, in certain cases, the phages are able to inhibit the development of phytopathogenic bacteria in the field and not only *in vitro*. Thus, in certain soils, lucerne sickness is caused by a bacteriophage which impedes the formation of root nodules and hence the nitrogen fixation (e.g. Demolon and Dunez, 1935); this phage can be demonstrated in the nodules, although only in those of approximately 3-year-old plants, as well as in the soil.



FIG. 208. The influence of expressed sap from tubers of *Orchis incarnata* on the hyphae of a mycorrhizal fungus. 1 Control: ordinary water. 2-5 Influence of sap. 2 After 6 hours. 3 After 22 hours. 4 After 2 days. 5 After 4 days. Approx. $\times 700$. (After Burges, 1939.)

Further, there is no doubt that, in some bacterioses, older diseased tissues contain a phage more or less specific for the bacterium concerned (e.g. Chester, 1933; Duyfjes, 1935; Kent, 1937, for the galls of *Bacterium tumefaciens*). Coons and Kotila (1925) isolated from rotting carrots a phage polyvalent for *Bacillus carotovorus* (soft rot of root crops), *Bacillus atro-septicus* (black leg of potato), and *Bacterium tumefaciens*.

Finally, there is no doubt that bacteriophage also occurs in healthy tissues surrounding the tumours of pelargonium and sugar beet, in healthy parts of gall-bearing tomato plants, and, finally, in a small percentage of healthy sugar beets that have been grown in *B. tumefaciens* contaminated soil. On the other hand, bacteriophage has never been found in plants which have had no contact with the pathogenic bacterium concerned (Chester, 1933; Kent, 1937).

The genesis of the phage is, however, still uncertain. Three possibilities exist. (1) The phage, in minute amounts, may have been introduced into the host plant from the initial culture. In this case, the phage must become enriched while in the diseased tissues. This appears, in fact, to happen; if, for example, a phage-containing culture of *B. tumefaciens* be injected into pelargonium, *Ricinus*, &c., and the phage be then re-isolated from the young tumours, it reacts more vigorously upon the initial culture than the original phage does after the same number of transferences (Duyfjes, 1935). Therefore, either it must now occur in larger quantities, or its virulence must have been enhanced while in the plant. (2) The phage may be produced by the bacteria themselves under the influence of their disintegration by the host plant. In this case, strains of phage from hosts of differing susceptibility may show differential virulence; this question has, apparently, not yet been closely examined. (3) The phage may be a defence product formed by the host plant. The first eventuality is the most probable but it is not easy to prove. It would be essential to work with indisputably phage-free cultures, if such exist, or to eliminate any phages present by treating the cultures with a reliable phage antiserum.

Lastly, it is doubtful if the phage is able to function in the plant tissues. If, for example, a *B. tumefaciens* phage be drawn up through the cut ends of twigs, or if it be hypodermically injected, the susceptibility of the given tissues to crown gall is not reduced (Kent, 1937). Similar observations have been made with carrots, as mentioned above. The phage, therefore, must come into direct contact with the bacteria prior to infection, and must be adsorbed by them to become effective; inside the plant tissues no adsorption appears to take place.

Fundamentally, therefore, nothing is certain concerning bacteriophages as a factor in the study of plant immunity: whether they are functional within the cells, whether they are formed by the bacteria under the influence of the host during the process of infection and in connexion with it, or whether they should not rather be considered as foreign substances which reach the host-parasite system only as contaminations.

dd) Acquired Virus Antibodies as a Factor in Anti-infectious Defence

Virus research, as a result of its special experimental possibilities, has provided a new approach to the study of plant immunity; thus, in mosaic-diseased tobacco, the presence of a true, induced antibody has been established *in vitro* for the first time, and its chemical properties fairly accurately determined.

The salient features of this work are as follows. The mosaic virus of tobacco is present in lower concentration in the expressed sap of resistant varieties of tobacco than in that of more susceptible varieties; thus its increase in resistant varieties must be hindered. This inhibition increases in the course of a single disease; thus, the virus concentration in a susceptible variety rises during the first 44 days after infection and then falls in the ensuing 29 days: hence there may be a gradual development of new virus-neutralizing defence substances. The existence of such an inhibiting substance was confirmed by Melchers and Schramm (1940) in the following way. If the sap from healthy and diseased tobacco plants, from which the virus protein has been extracted by the ultra-centrifuge, are each mixed with the same quantity of virus protein (e.g. 10 μ g. virus protein to each 1 c.c. sap) then the juice from sick plants is only half as effective as that from healthy ones; half of the virus protein has thus been inactivated by the antibodies produced in the diseased plants. Chemically, the neutralizing substance, in contrast to antibodies in the animal kingdom, is not a protein but a heat-resisting compound of low molecular weight and soluble in butanol.

Besides such direct proof, there is also indirect evidence for the existence of acquired antibodies against plant pathogenic viruses. For instance, what is the explanation of the fact that tobacco mosaic immunizes against aucuba mosaic (p. 313)? The most immediate explanations are that all the virus-attainable points in the host cells have been occupied by the primary virus and are, therefore, closed to a following secondary virus, or that all the substances essential to the building of proteins have been used up by the first virus. Yet these hypotheses are inadequate: if a mixture of the two viruses be inoculated into tobacco plants, they segregate in the host cells so that in any given tissue lesion only one of the viruses is ever present; a given host cell or group of cells always contains only one virus. Far more complex processes, therefore, must take place in the host cells than the mere saturation of points of attachment or the using up of native proteins.

Further, premunizing activity is limited specifically or group-specifically, hence, it does not hold good for all viruses; it extends several cell layers beyond the directly infected zone and includes cells whose points of attachment cannot be saturated or whose protein supply cannot yet be exhausted. The question is still open, whether the antibodies formed in the immediately diseased cells can diffuse outwards for a distance of seven cell layers into the still healthy tissues, or whether pathological metabolic products

are able to diffuse this distance and there initiate the formation of new antibodies.

Another example of indirect evidence for the production of anti-infectious substances protective against viruses is afforded by the Californian curly top disease of sugar beet (Wallace, 1939). If a tobacco plant be inoculated with the virus of this disease, it recovers after a certain time but remains a virus carrier. If the virus from a recovered plant be transmitted by the cicada, *Eutettix tenellus*, to healthy tobacco plants, the disease develops more vigorously than when healthy shoots are grafted on to recovered virus-carrying plants. This can be explained on the assumption that, in the graft, antibodies as well as virus pass from the stock into the scion, whereas the insect transmits only the virus.

2. Antitoxic Defence Reactions

As we have seen above, anti-infectious defence reactions, even under favourable conditions, are able at most to weaken and localize but not to eliminate parasites; hence a local infection persists. In addition to the direct struggle against the parasite, there accrues to the host plant the new task of protecting itself against the pathogenic metabolic products emanating from the focus of infection, in particular, against the toxins of the parasite and the necrogenous products of disintegration of its own injured cells and tissues. This protection is afforded by the antitoxic defence reactions (p. 278). In contrast to the anti-infectious reactions, their aim is not to avert infection but to avert disease. As in man, so in plants, not all foreign substances are able to release such reactions, but only those which have an antigenic action; other substances can inundate the plant without evoking counter reactions.

Whilst the anti-infectious defence reactions in human medicine are associated with the names of Pasteur and Metchnikoff, the antitoxic reactions are associated with that of Ehrlich. This investigator started from the hypothesis that the human body responds to the stimulus of a pathogenic agent by the formation of specific antitoxins (receptors), which possess 'side chains' analogous to the side groups of the benzene ring, and by means of which the pathogen is poisoned or neutralized, i.e. becomes chemically bound and, therefore, harmless. The body is thus protected against a fresh disease as long as the corresponding antitoxin in the blood does not fall below a certain value.

In addition to this biochemical protection by antitoxins poisonous to the pathogenic agent (i.e. which put it out of action) the human body also possesses histogenic protection through its demarcation tissues, which localize pathogenic activity and often the pathogen itself. This accessory anti-infectious effect becomes obvious if, for example, an encapsulated tuberculous infection becomes inflamed and active again months or years later. The two kinds of human demarcation reaction which are of present interest lead either to localization and elimination of the injurious focus

(e.g. metastatic abscesses) or only to localization by the formation of a permanent protective wall (e.g. tubercle formation).

If a pyogenic focus (pyaemic metastasis) arises anywhere on the circulatory route, the first symptom of bacterial action observed in the human body is injury to the tissue cells with a rapid appearance of leucocytes (white blood corpuscles) which emerge from the enlarged capillaries. These blood corpuscles probably work mainly through their enzymes which dissolve the bacteria and the disintegrating tissue fragments. Other blood cells, lymphocytes and plasma cells, surround the leucocytes and probably absorb and carry away the products dissolved by them.

These two first zones, the leucocytic and the plasmocytic, serve to disintegrate the noxious agent and, therefore, can be regarded as a living protective wall (phagocytosis), corresponding to front-line soldiers. Fairly soon, a zone of fibroblastic connective tissue resembling a permanent fortification arises which, finally, becomes transformed into a non-nucleate fibrilloid capsule. This non-specific insulating tissue delimits the lesion from the healthy organ tissue and protects the latter from contact with any injurious products. Later, the ring thickens until, ideally, there remains only a small homogeneous area of connective tissue (the scar).

In certain cases of tuberculosis the tubercle bacilli stimulate the formation of specific tubercular granulation tissue. This is a zone of varying breadth, depending upon pathogenicity and resistance and formed of specialized connective-tissue cells, the so-called epithelioid cells, which obviously serve directly in the defence of the body against tubercle toxins and bacilli and, therefore, have an antitoxic as well as an anti-infectional character. The central focus of disintegration and the defensive shield of epithelioid cells together form the 'tubercle'. In favourable cases, the dead central region becomes calcified but only after several years does the production of new epithelioid cells come to an end, evidently because there is no longer any stimulus from diffusing bacterial poisons; only then does the connective tissue ring directly abut on the calcified portion, a sign that the lesion has ceased to be infectious.

Protection of the organism depends, therefore, in both cases, upon the possibility of an encapsulation of the infection focus occurring at the right moment (time factor); it is a kind of race between the body's tendency to encapsulation and the pathogen's ability to spread and cause injury. Should the micro-organism win, the infection is taken into protective custody: in the case of purulent metastases it is eliminated, whereas in the case of tubercle formation it is merely isolated; if the micro-organism loses, the disease spreads farther, enters the blood-vessels, and produces secondary foci at a distance.

In the infectious diseases of plants, relatively little is known about anti-toxic defence reactions. It is clear that antigenic activity can, as in human diseases, arise from the metabolic products of the pathogen as well as from those of the body's decomposition. A direct antigenic activity of the pathogen occurs, for instance, in those bacteria and fungi which cause

wilting diseases, 'apoplectic' phenomena, galls, &c.; the chemical nature of the toxins concerned is, as yet, little understood (p. 243). However, as in human pathology, antigenic activity may derive just as frequently from necroses as from the pathogen which causes them.

In both cases we must assume that the protoplasm of the host tissue somehow resists the pathogenic products, and attempts to destroy and eliminate them. It would contradict all our notions of living things if they allowed themselves to be injured every time without reacting. However, nothing is known about this plasmatic defence or about specific plant antitoxins.

At present, our knowledge is limited to the second group, i.e. the numerous demarcation or delimitation reactions, which occur in addition to the hypothetical plasmatic defence reactions, and frequently localize the harmful agent to the benefit of the whole organism. The effect of this demarcation reaction is similar to that of the anti-infectional defence reactions (p. 289); the pathogen is not killed but merely localized. The similarity of their effects makes it far from easy, in some plant diseases, to distinguish sharply between the two reaction groups. Nature knows nothing of our division of her reactions into antitoxic and anti-infectional groups; analytic categories are abstractions of the human mind. As in human medicine, a given demarcation reaction, in addition to its primarily antitoxic character (e.g. encapsulation of the toxin by a special cell barrier), may have a secondary anti-infectional effect (e.g. prevention of the further extension of the parasite).

Because these primarily antitoxic and only secondarily anti-infectional barrier reactions do not directly combat the parasite (in that they do not attack and injure it directly), but only indirectly prevent its spread by putting a new obstacle in its way, they have been described repeatedly in the literature as newly formed local factors of induced resistance. Naturally such terminology is as 'correct' and as 'incorrect' as any other; it is only a question of mental economy, of how best to lighten the task of understanding. According to our terminology, resistance depends on innate, pre-infectional, anti-parasitic barriers (p. 251). According to this way of thinking, an induced resistance arising subsequent to infection is a contradiction in terms; in our classification, the post-infectional production of demarcations, induced by the parasite, is a defence reaction (as it is in human pathology).

According to their degree of activity, plant demarcation reactions, like anti-infectional processes, are either normergic or hyperergic (Table XLVI). As an example of the former, the predominantly histogenic demarcations will be considered; as an example of the latter, the predominantly gummous demarcations (p. 341).

(aa) Histogenic Demarcations as a Factor in Antitoxic Defence

Delimiting tissues of predominantly antitoxic character may be produced in leaves, branches, and roots (p. 336).

A leaf example is the shot hole effect in stone-fruit trees (Fig. 209). If the leaves of *Prunus* spp. are infected by the conidia of *Clasterosporium carpophilum* the causal agent of shot hole disease, the germ tubes penetrate either through the epidermis or through the stomata into the leaf interior, and proliferate in all directions in and between the cells. Thirty-eight hours after infection no change can be observed macroscopically, but microscopically (see Fig. 210, 1), the guard cells of the stomata through which the parasite entered are seriously injured and about to collapse and their walls are thickened and brown. In consequence of the distant action of the parasite's toxins, the nearest hypodermal cell (Fig. 210, 1), although not directly attacked, shows brown discoloration (hyperergic effect). Moreover, in other neighbouring cells, the chloroplasts have disappeared and, farther away still, they have shrunk and become light green. On the other hand, in the spongy mesophyll no changes are yet apparent beyond a darker colouring of two cells (again the result of action at a distance).

Fifty-six hours after infection the lesion is already 1–2 mm. wide and, therefore, visible macroscopically. The mycelium has invaded the spongy tissues (Fig. 210, 2) and the neighbouring vascular bundles in which it has obliterated the phloem parenchyma. A few epidermal cells are brown and collapsed. The hypodermis is disorganized and collapsed so that the infection spot appears on the leaf surface as a small pit. Numerous cells of the spongy mesophyll are in process of degeneration and the toxins of the parasite are causing disintegration of the chloroplasts right up to the palisade tissue.

After 4 days the hyphae have reached the palisade tissues (Fig. 210, 3). Macroscopically the necrotic cells in the centre of the lesion are seen as a brown patch, surrounded by a lighter green zone (the cells containing the disorganized chloroplasts).

Histogenic defence reactions set in between the fourth and eighth days. At a distance of about 20 cells from the brown necrotic zone, a narrow band of cells which involves all the leaf tissues begins to swell slightly; the cells are squeezed closely so that the intercellular spaces disappear, as do the chloroplasts; the protoplasm becomes denser, the nuclei increase slightly in size, and the cells assume a meristematic character. In consequence of the disease, tissues which had already matured have again become capable of undergoing division; their correlations have been broken down and thus the corresponding growth hormones have been formed or

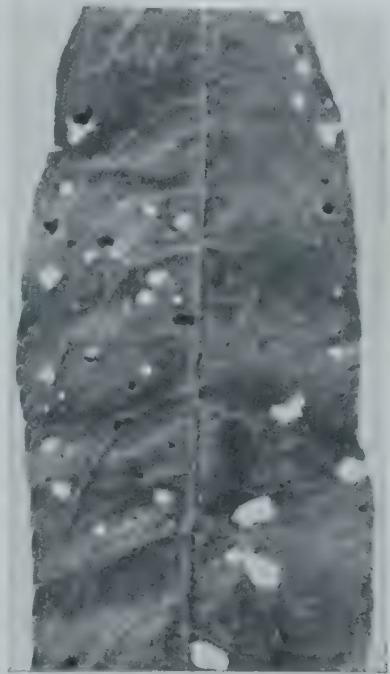


FIG. 209. Shot hole effect on peach leaves, caused by *Clasterosporium carpophilum*. A few, dark-coloured infection loci are about to be eliminated. Nat. size. Original.

mobilized (perhaps necro-hormones in Haberlandt's sense). Fig. 211, 1 shows such a secondary meristem in an 8-day-old infection; the cell walls are thin in contrast to the thickening and lignification of all the cell walls in the infection zone. Vigorous cell division also sets in throughout the vascular bundles, whose parenchyma cells have been rejuvenated, and the xylem elements are squeezed to the periphery of the bundle.

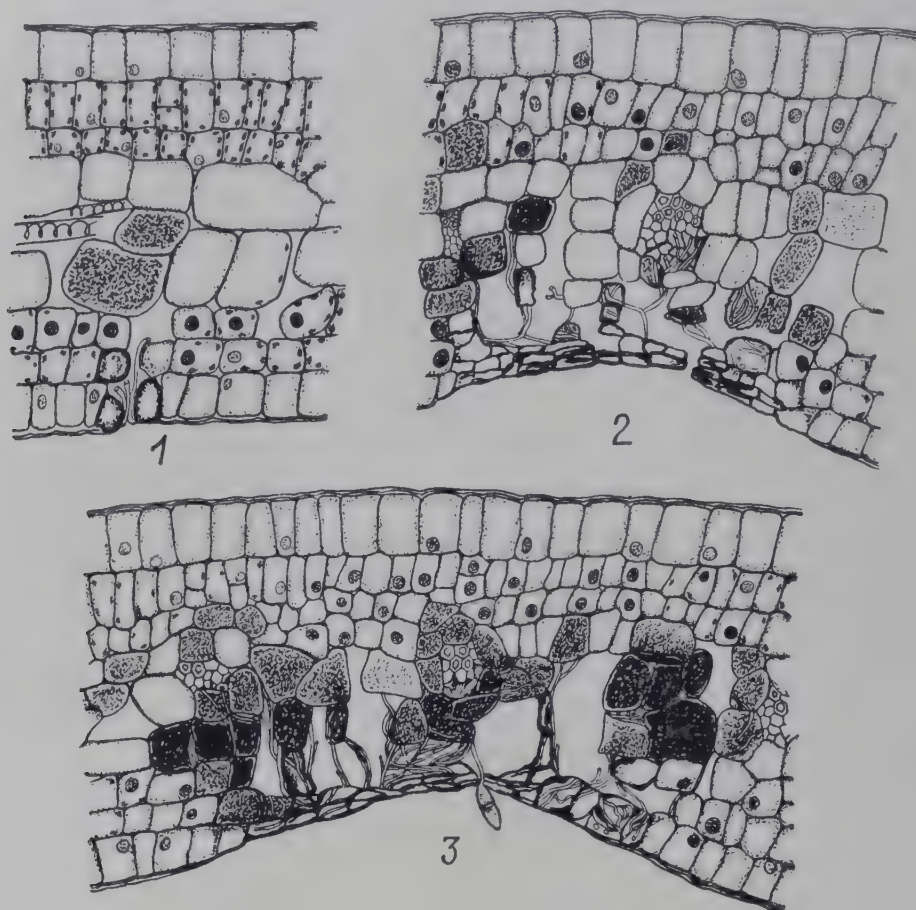


FIG. 210. The infection of leaves of cherry laurel (*Prunus Laurocerasus*) by the pathogen of shot hole disease, *Clasterosporium carpophilum*. 1 Infection locus after 38 hours. 2 After 56 hours. 3 After 4 days. $\times 215$. (After St. Roth; from Gäumann, 1944.)

On the one hand, this abscission tissue interrupts the supply of nutrients from the healthy tissues to the lesion (so that this gradually shrivels) whilst, on the other hand, it protects the healthy tissues from damage by the toxins of the parasite and by the necrogenous products of the dying lesion.

In young leaves, beginning at the lower epidermis, the middle lamellae in a certain zone begin to disintegrate, usually in such a way that one cell row accrues to the focus, while the three and four others remain part of the healthy leaf. In this way, the focus of infection becomes spatially cut off from the healthy tissues and subsequently is actively thrust away from the lower side of the leaf.

Scarring now begins along the edge of the wound by the initiation of a wound periderm. A middle cell row, the phellogen, forms on its outer side two or three slowly lignifying and then suberizing layers of phellem cells and, on the inside, three or four rows of only cellulose thickened phello-

derm (Fig. 211, 2 and 3). With this process the histogenic demarcation reaction is completed.

Similar secondary abscission tissues, which delimit and scale off the lesion, are also formed on branches under the influence of the parasite (Fig. 212).

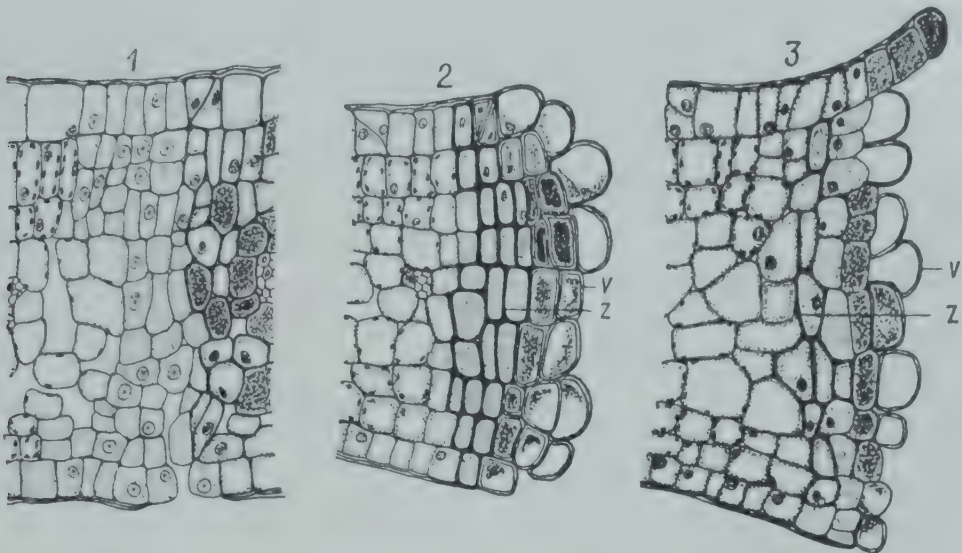


FIG. 211. Demarcation and elimination reactions in shot hole disease. 1 Induced meristem about 20 cells distant from necrotic zone (always on right-hand side). 2 and 3 Scar reaction by lignification and suberization of the outermost cell layers. Z. Thickening with cellulose. I. Lignified thickening. $\times \frac{2}{3}$. (After St. Roth; from Gäumann, 1944.)

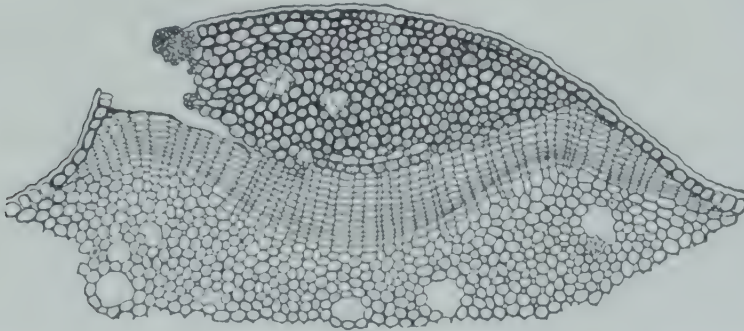


FIG. 212. A section through a scab on a branch of almond (*Prunus Amygdalus*), caused by *Clasterosporium carpophilum*. $\times 60$. (After Samuel, 1927; from Gäumann, 1944.)

In *Prunus* spp. the sequence of defence reactions against *Clasterosporium carpophilum* is, therefore, carefully adjusted (*planmässig*), to ensure the preservation of the whole. In this simplified presentation, the shot hole effect is due to two components, a delimiting and an expulsive reaction. The former leads to the initiation of a secondary meristem and, through this, to the production of an abscission layer which, in the leaf, extends from epidermis to epidermis, thus insulating the lesion together with the metabolic products of the parasite and the disintegration products of the dying host cells. In the leaf the eliminating reaction ends when the lesion is expelled and in the twig when it is scaled off. In the cherry, for instance, because of the dropping out of the lesions, the foliage appears as if it had

been riddled by shot; hence the name of the disease. Following the elimination a scarring reaction ensues.

This chain of reactions is not initiated directly by the parasite but by the necrosis which it causes. The demarcation reaction is non-specific to the extent that the genesis of the necrosis is a matter of indifference, whether, for example, it be due to necrogenous products from body wounds, toxins from the parasites (Fig. 213), or any other organic or inorganic foreign poisons (e.g. fungicides).



FIG. 213. Shot hole effect on leaf of cherry laurel (*Prunus Laurocerasus*), induced by an injection of a mycelial extract of *Clasterosporium carpophilum*. Nat. size. Original. E. Böhni.

In the first place, therefore, the shot hole effect is not directed against the invading parasite but against its toxins and the host's own necrogenous products; thus it represents primarily an anti-toxic defence reaction. In consequence, the demarcation layer does not provide an unconditional protection against parasites, since these can, in certain cases, grow through the barrier. As a rule, however, the check is sufficiently great not only to dam up the parasitic and necrogenous toxins, but (in the guise of a newly formed inhibitory zone) to hold up the parasite, and thereby localize the disease focus; thus, the antitoxic immunity reaction has a secondary anti-infectious action.

The mechanism of the limiting reaction is as non-specific as its cause. It corresponds to the fact that, throughout the plant kingdom, an abscission or delimiting meristem (dehiscence tissue, scarring reaction, &c.) is commonly induced by local injuries. In the present case the parasite causes the necrosis, this gives rise to

necrogenous substances, and these, in the usual way, stimulate the formation of wound periderm.

In spite of the non-specificity of the cause and means, the shot hole reaction is specific for *Clasterosporium carpophilum*, in so far as it is just this fungus which is markedly toxic to *Prunus* tissues and gives rise to local necroses, which, in turn, initiate the sequence of reactions described, terminating in the cicatrization of the lesion. The clinical picture of local, encapsulated lesions which finally drop out is, therefore, diagnostic of shot hole disease and recognizable as such.

Other fungi, e.g. *Tranzschelia pruni spinosae* (rust of plum trees), possess no such antigenic activity and, therefore, do not give rise to any delimiting tissue on *Prunus* leaves. Still other fungi, e.g. *Gloeosporium fructigenum* (bitter rot), possess fully developed antigenic properties (the effect of their mycelial extracts is as shown in Fig. 213) but they develop more rapidly than the demarcation reaction can follow, so that this lags behind; the

race (see p. 329) between pathogen and host is here won by the pathogen. In both cases the result is that the parasite successively colonizes the whole leaf. It is not single elements or components of the course of the reaction that make the shot hole effect a recognizable and specific antitoxic defence reaction, but their relations to one another, their totality.

In other diseases of plants the demarcation tissue in the leaves does not extend from epidermis to epidermis but develops periclinally to the localized focus of infection (Fig. 214), and leads to scabbing.

These delimiting and eliminating reactions correspond, *mutatis mutandis*, to the earlier described response of the human body to purulent meta-

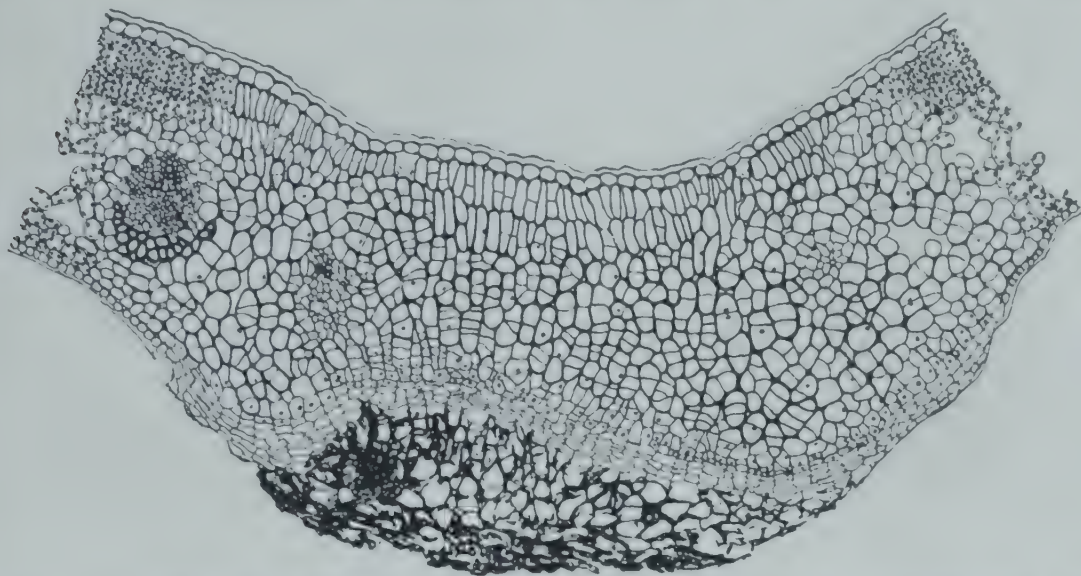


FIG. 214. Sloughing of a localized infection caused by *Sphaceloma Fawcettii*, on a leaf of *Citrus limonia*. $\times 115$. (After Cunningham, 1928.)

stases. In plants, however, the analogue to tubercle formation occurs more frequently, i.e. only the first stage of the shot hole effect, the delimiting reaction, occurs but not the expulsive or scabbing reaction. The necrotic, encapsulated, and withered tissues remain connected to the healthy parts and perforation of the leaves does not take place. This happens in numerous leaf spot diseases, where each spot corresponds to a focus of infection which has been encapsulated by the localizing reactions of the host; e.g. in leaf spot of sugar beet, caused by *Cercospora beticola*, and that of ivy (*Hedera Helix*) caused by *Phyllosticta hedericola* (Fig. 215). The corky demarcation tissue, if viewed against the light, appears as a thin, dark-brown line surrounding the lesion; this distinguishes parasitic lesions from chronic injuries due to industrial gases which, so long as actual burning does not occur, do not cause such demarcations but more diffuse chlorotic effects without marked defence reactions in the host.

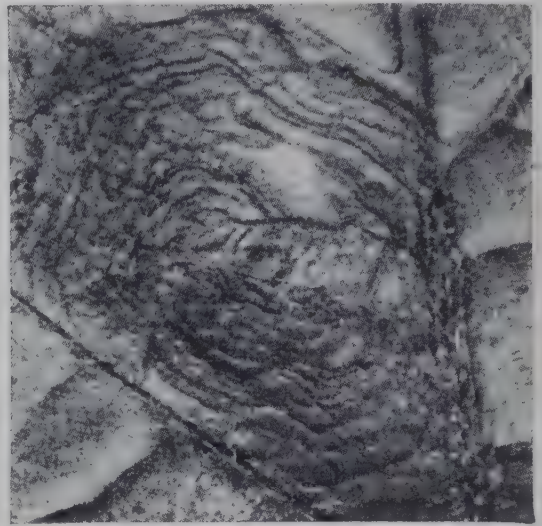
How slight, on occasion, the parasite-inhibiting (not toxin-inhibiting) effect of demarcation tissue can be is shown in two potato diseases; thus in leaf spot (Fig. 216) and in common scab of the tubers (Fig. 217) the corky barriers are continually being penetrated by the pathogen and, therefore, form irregular concentric rings, one outside the other.

Examples of demarcation reactions in branches and roots, sometimes accompanied by an expulsive or scaling reaction, have already been met with in the shot hole disease (Fig. 212), in mistletoe infection (Fig. 206), and in potato wart disease (Fig. 191). Three further examples will now be discussed rather more thoroughly: root rot of tobacco, *Armillaria* root rot of deciduous and coniferous trees, and the natural cleansing of branches of woodland trees.

The occurrence of root rots or foot rots of tobacco (Fig. 218) caused by *Thielavia basicola* is greatly influenced by soil temperature; in the



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FIG. 215. *Phyllosticta hedericola*. An encapsulated, dead, and in part already bleached infection locus on an ivy leaf. Suberised demarcation tissue is dark brown. Nat. size. Original.

FIG. 216. Leaf spot disease of potato leaves, caused by *Alternaria solani*, showing concentric antitoxic demarcation zones. Approx. $\times 8$. (After Maag, 1944.)

vicinity of $17-23^{\circ}\text{C}$. the disease is frequent, while between 26° and 32°C ., even with the heaviest soil contamination, it is practically absent. Since, however, the optimal temperature of both the fungus and the host plant lies between 28° and 32°C . and the parasite shows only little growth below 23°C ., the high incidence of disease at lower, and the slight infection at higher, soil temperatures must be due to a special thermally conditioned relationship of the host. This is manifested histologically in the events of suberization (Conant, 1927).

Thielavia basicola is a markedly toxin-producing parasite: the cork reactions against it are primarily antitoxic and only secondarily anti-infectious. The efficiency of the cork layers against the fungal hyphae is correspondingly limited; single growing hyphae are unable to penetrate suberized cells but, in mass infection, the fungus breaks through the cork layers and makes its way into the interior of the root. The time at which the cork layers are formed and the stimulus to their production no longer coincide.

Among the pre-infectious processes of suberization which arise naturally

and have no connexion with the parasite, the formation of periderm by the pericycle is the most decisive since it results in the production of a closed mantle of cork around the vitally important vascular cylinder. Roots which have already discarded their corky epidermis but in which the suberization of the pericycle has either not begun or is not sufficiently advanced run the greatest risk of infection. This danger is slight in the resistant varieties (e.g. Little Dutch) because, in them, the cell division in the cork cambium, even at low temperatures (20°C.), begins early and occurs together with secondary growth in the roots (in consequence of which the corky epidermis is ruptured and thrown off) whereas, in the susceptible varieties (e.g. Maryland Broadleaf and Susceptible Burley), suberization takes place only after the epidermis has been lost.

The fact that, in susceptible varieties, periderm is laid down relatively late at low soil temperatures affects also the initiation of the lateral roots; they arise in the pericycle and break through the primary cortex, so that this is torn in their vicinity and for a time they are readily accessible to infection. On the other hand, at high soil temperatures, even in the susceptible varieties, a closed protective cork mantle is formed on the inner side of the primary cortex because, at these temperatures, the periderm is laid down simultaneously with the lateral roots.

If these pre-infectional resistance barriers and any other innate powers of inhibition fail, the parasite invades the cortical tissues and its toxins diffuse out in advance of it. In response, in the resistant varieties of tobacco at all soil temperatures, new cork layers are initiated in the cortex or in the pericycle and insulate the focus of infection. In susceptible varieties, these new post-infectional cork layers, like the normal periderm at soil temperatures of 20°C. , are formed so slowly that, at first, no histological resistance is opposed to invasion by the fungus or to its toxins; only at $25\text{--}30^{\circ}\text{C.}$ is the new cork layer beneath the site of infection formed quickly enough to lead to encapsulation of the infection and, consequently, to a diminution of attack.

Thus, at low soil temperatures, in susceptible varieties of tobacco, both the natural pre-infectional suberization (resistance factor) and the induced

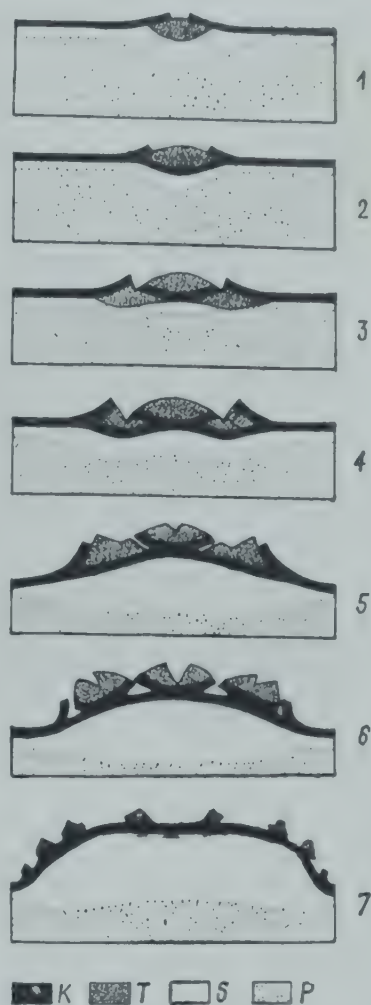


FIG. 217. Diagram showing development of a scab (umbo-nate scab) on a potato tuber. The pathogen, *Actinomyces scabies* in stage 1 has penetrated via a lenticel (Fig. 50). K epidermis and wound cork. T dead parenchyma. S starch-free parenchyma. P starchy parenchyma. (After Noll, 1939.)

post-infectious suberization (antitoxic defence reaction) take place too slowly. Hence, in spite of temperature levels unfavourable to the parasite, the plants receive insufficient protection; in consequence they become diseased. On the other hand, resistant varieties act and react within this temperature range with a speed which the susceptible varieties attain only at high soil temperatures.

As in the *Prunus* leaves, so in the tobacco roots the induced histogenous demarcations are not something fundamentally new. No new capacities of the individual are concerned in them; the natural pre-infectious and induced post-infectious periderm formations are histologically similar and,

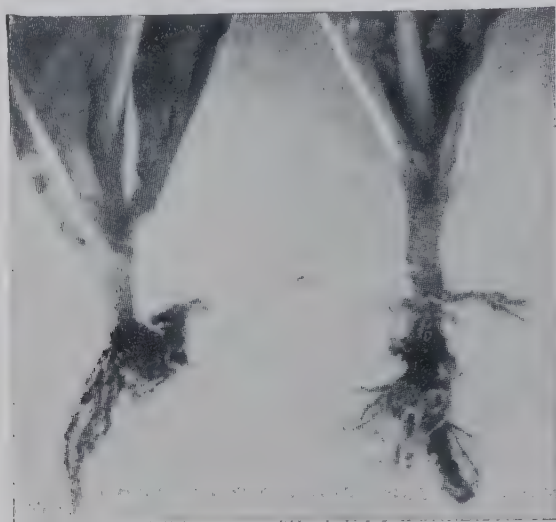


FIG. 218. Symptoms caused by *Thielavia basicola* on tobacco.
(After Sattler, 1936.)

therefore, their antitoxic and anti-infectious characteristics probably also correspond.

However, their status in the individual as a whole is different. The pre-infectious periderm formations arise in special places at a particular time in the development of the individual and are characteristic for the given variety. On the other hand, in the case of the post-infectious periderm, the natural co-ordinations of the individual are disorganized; tissues which have already matured become rejuvenated in response to the diffusion into them of the toxins of the parasite and form adventitious periderm in unusual places and at irregular times. These adventitious periderm formations have essentially the same structure as those arising naturally, but whereas the capacity for excluding toxins is only a subsidiary effect in the case of the natural periderm, which is primarily concerned with quite other physiological and mechanical functions, in the case of the induced periderm it returns 'teleologically', to the centre of the picture; it is their primary function and since, for example, their mechanical functions have been abandoned, it appears to be the only valid reason for their genesis. They are characteristically effective only against the toxins of the pathogen, i.e. they are exclusively 'directed' against them.

Histologically the primary and the induced periderms resemble one

another, but the place and time of their genesis and their significance for the individual concerned are different.

Similar corky delimiting tissues and, to some extent, dehiscence tissues, occur, for example, in the roots of flax varieties resistant to flax wilt (*Fusarium lini*).

In the second example to be discussed, the attack on young roots of deciduous and coniferous trees by the honey agaric (*Armillaria mellea*), a similar chain of delimiting and scaling-off reactions takes place as in the root rot of tobacco. If attacked by a rhizomorph, resistant trees form a secondary cork cambium (Fig. 219, *c*) which enables them to localize and eliminate the disease focus. This secondary cork cambium is again nothing fundamentally new since its formation usually occurs with the scaling-off of the primary bark which would take place normally a year or so later but which is now formed prematurely under the influence of the parasite.

The secondary periderm, as in the case of the periderm of tobacco, serves only secondarily as a mechanical barrier; like the primary periderm (Fig. 45) it can be actively broken through, on occasion, by the rhizomorphs. Its primary significance is antitoxic. The honey agaric is extremely poisonous and often, even from one local focus of disease (e.g. on the roots), is able to damage or kill the whole tree. The induced periderm, therefore, is primarily a chemical barrier against the diffusing toxins of the parasite and prevents the underlying tissues from becoming poisoned and having their resistance weakened.

Probably similar conditions obtain in the attack on chestnuts by *Endothia parasitica* (Bramble, 1936).

Finally, the histogenic, antitoxic, demarcation reactions are of special economic importance in the third example, the natural branch casting of forest trees. In order to produce wood of good quality a forester seeks to cultivate the stand in such a way that the trees themselves automatically shed their surplus under-developed branches. The dead branches become

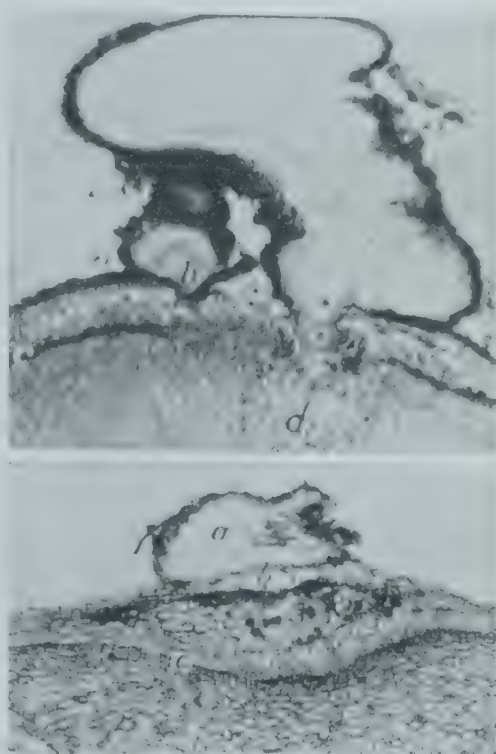


FIG. 219. Antitoxic defence reactions against the honey agaric (*Armillaria mellea*). 1 The mycelial strand *a* has penetrated into the xylem of the root of a pear tree, *Pyrus communis*, and at *d* has produced a local focus of disease. The mycelial strand *b* is breaking through the original periderm and penetrating the primary cortex. The layer *c* represents newly formed secondary periderm which cuts off the affected cortical parenchyma. 2 Root of a Californian walnut (*Juglans Hindsii*) in which the infection loci due to the rhizomorphs *a* and *b* are encapsulated by the new cork layer *c*. Approx. $\times 28$. (After Thomas, 1934.)

attacked and gradually destroyed by wood-rotting fungi (usually weak parasites) such as *Polystictus versicolor*, *Stereum purpureum*, *Polyporus vaporarius*, and *Coniophora cerebella*. As a reaction to the diffusion of the necrogenous substances, in favourable cases, the base of the branch cuts itself off by means of a gummous protective tissue (Fig. 220) and later, after the fall of the branch, it becomes occluded from the sides without scarring, i.e. the wound heals evenly.

In this case also, the antigenic effect is not primarily due to the fungus, although culture filtrates of *Stereum purpureum* are very toxic (Brooks and Moore, 1926), but to the necrogenous substances formed in the infected

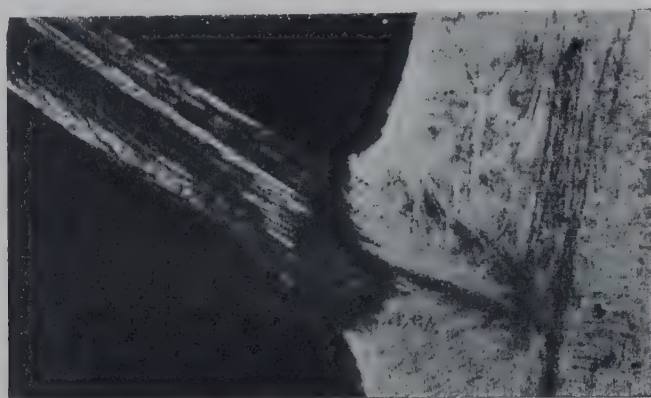


FIG. 220. Section through the base of a dead beech branch showing the dark brown demarcation layer. As a rule it extends from the cambium on the upper side of the branch right through the branch to the pith, proceeds in the pith a short way towards the trunk, and then curves back to cambium on the under side of the branch. Approx. $\times \frac{1}{10}$. (After Gelinsky, 1933; from Gäumann, 1944.)

branches. Thus the effect is primarily one of protection against self-poisoning due to local necroses. In addition, these demarcation tissues have an anti-infectious significance, like the tissue wall in human pathology mentioned earlier. As a rule parasitic fungi are able to penetrate them only whilst they are being formed and not later.

Where natural branch casting, i.e. spontaneous biological shedding of dead branches, does not take place, the results of the damming up of the necrogenous substances and, eventually, of the fungus itself, by the basal delimiting layer, are different for conifers and deciduous trees.

In conifers, even where infection of the trunk by wood-rotting fungi does not occur, the stumps of the dead branches persist as foreign bodies in the subsequent annual rings. There is little or no occlusion and later, owing to their marked shrinking, they become horny knots which fall out of the cut timber and reduce its value.

In deciduous trees, especially the beech, either the necrogenous substances diffusing into the trunk cause effects similar to the hyperergic reactions mentioned above, or the fungi find a starting point in the dead branches from which to attack the trunk. In both cases the technical value of the trunk is reduced.

(bb) Gummous Demarcations as a Factor in Antitoxic Defence

As in the normergic plasmatic defence reactions, so in the normergic demarcation reactions their efficiency is limited. The encapsulating effect described above is achieved only in certain infectious diseases, and then often only under favourable environmental conditions. In numerous other cases the histogenic demarcations fail (either because their efficiency is too low or they appear too late) and the pathogenic agent, therefore, begins to overrun the organism. But in certain infectious diseases there appear in the organism new, hyperergic demarcation reactions which (like the



FIG. 221. Sealed-off, infected beech branch, which provided the stimulus to hyperergic gummous reaction. The reaction zone in the centre of the trunk, which appears dark grey in the picture, is really reddish-brown.

Approx. $\times \frac{1}{5}$. (After Tuzson, 1905; from Gäumann, 1944.)

hyperergic anti-infectional defence reactions) partially compensate for the inefficiency of the normergic reactions.

For example, if, in the beech, the histogenous demarcation (see Fig. 220) proves inadequate and the necrogenous substances reach the trunk, the mature wood of a certain age (40 years or more) reacts very intolerantly to their stimulus with a pathological heart-wood reaction, red or false heart (Fig. 221), which contrasts with the naturally occurring 'true' heart of the oak. This morbid heart-wood reaction is caused in beeches and in many other deciduous trees by local infection of the trunk, &c. (Fig. 222).

The red heart is produced by a combination of hyperergic, histogenic, and gummous demarcation reactions. In the hyperergic reactions, tyloses arise in the medullary ray cells over a distance of several metres, and project into the vessels, thus blocking circulation in the mature wood and causing the slow death of the entire reaction zone. In the gummous reactions in this zone tannin-like heart-wood-forming substances are produced in excess in the cell walls, in the medullary rays, and in the cells of the wood parenchyma. These substances are oxidizing tannin

compounds which, when treated with sulphuric acid, belong to the lignin fraction, but in contrast to this are not methoxylated. The histogenic and gummous barrier produced in this way cuts off the disease focus from the sound tissues and protects the organism from being flooded by the toxic necrogenous substances (protection from self-poisoning).

These hyperergic, antitoxic demarcation reactions, like the normergic, are non-specific in so far as the nature of the stimulus to the formation of the necrogenous substances is immaterial. Similar reaction chains can be produced in the beech by frost damage (temperatures under -30°C.) (frost heart; Jahn, 1931; Larsen, 1943).

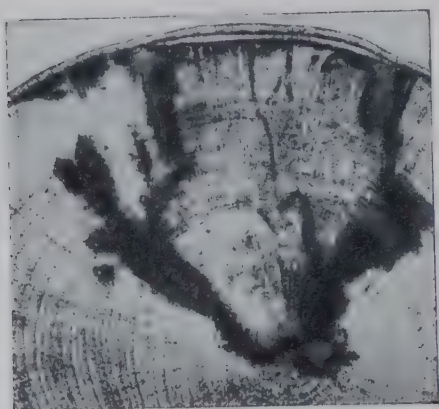


FIG. 222. Antitoxic defence reaction of a beech trunk artificially infected with *Stereum purpureum*, $3\frac{1}{2}$ years after infection and 60 cm. above the point of inoculation. In the centre is the white rot focus of disease and around it the gummous reaction zone. The cambium has remained intact and its activity has been slightly stimulated. Approx. $\times \frac{1}{2}$. (After Münch, 1910; from Gäumann, 1944.)

Similarly, the component processes of the hyperergic, antitoxic demarcation reactions are non-specific. Gummous reactions also arise in other ways as we have already noted in wart disease of potato (Fig. 190), cereal rusts (Fig. 199), and branch casting (Fig. 220). Also the formation of tyloses can occur independently of fungal infection, and necrogenous substances can be produced as a result of the mere entrance of air into the vessels (Ebes, 1937).

Finally, the hyperergic, antitoxic demarcation reactions are also non-specific in their effect in so far as the newly produced barrier not only hinders the spread of the toxins and the necrogenous substances but also the attacking fungus itself, once the heart-wood has been formed (subsidiary anti-infectional effect). Hence, under certain circumstances the pathological heart-wood tissue is more resistant to fungi and more durable than normal mature wood.

But in spite of the fact that the cause, the means, and the effect are non-specific, all these reactions produce in their totality, their interreaction, and their spatial distribution, a characteristic, specific clinical picture, i.e. the syndrome of red heart of beech, as the product of hyperergic, antitoxic defence reactions by the still-living mature wood of the beech against necrogenous substances emanating from local infections.

The antitoxic effect of the hyperergic gummosis shows still more clearly in silver leaf of plum trees (Fig. 70).

In certain plum varieties the capacity for disease resistance depends on the readiness of the woody tissues to produce hyperergic gummosis (Brooks, 1928). The Victoria plum reacts only weakly and, therefore, falls an easy victim to the disease. On the other hand, the trunks of the Pershore variety do not become diseased. They respond to the stimulus of the pathogenic agent with an intense local gummosis and form, around the focus of in-

fection, a barricade of tannin materials and tyloses which encapsulates the poisonous substances as does the pathological heart-wood-forming zone in the beech (Fig. 222). The regular formation of such an antitoxic defence zone in the beech may, perhaps, be the reason why, in spite of its frequent attack by *Stereum purpureum* ('white rot') the tree does not develop silver leaf disease.

Both varieties of plum are susceptible to infection and, in both, local foci of disease arise, but the Pershore variety is disease-proof because it is able through its antitoxic defence reaction, hyperergic gummosis, to prevent the inundation and poisoning of its tissues by the pathogenic substances emanating from the local focus of disease.

The effect of the antitoxic defence reactions in man and in plants can be summarized as follows: man, in favourable cases, neutralizes (detoxicates) the toxins and the necrogenous substances, or localizes them by histological demarcation reactions: the plant has only the latter possibility at its disposal (localization of the pathogenic agent). In the same way, the antitoxic behaviour coincides with the anti-infectious behaviour, since man is equally able to destroy the pathogen in many infectious diseases, whereas the plant can only localize it.

3. *Induced Tolerance*

If a plant is unable to localize the pathogenic agent (the pathogen or its toxin) and consequently becomes flooded by it, its fate is not necessarily hopeless. In certain cases it can, as a new form of protection against disease, simply 'ignore' its existence. Its tissues may be overrun by the pathogenic agent (whether by the pathogen or its toxins is immaterial) but it 'takes no notice', it 'endures' the agent but does not react to it any more, it has become tolerant (induced tolerance, p. 280).

Induced tolerance has one feature in common with the antitoxic defence reactions, viz. that it obviates the consequences of infection but not, as the latter do, by neutralization or localization of the damaging substances, but rather by adopting the opposite extreme, by a desensitization of its own body, so that it no longer reacts in a perceptible way to the pathogenic agent and, therefore, does not become diseased.

In early times this biological phenomenon was exemplified in relation to the problem of immunity to poison (habituation to poison or conversion to tolerance). It is related of Mithridates VI, King of Pontus, 123 B.C., that he saturated his body with poisons and, therefore, became immune to them; hence the expression 'Mithridatism'. The education of young undergraduates might appear to be similarly directed to the production of immunity to alcohol!

In present-day medicine there is a hint of induced tolerance in the phenomenon of typhoid carriers. In many cases of typhoid fever, the clinical 'cure' (the disappearance of symptoms, i.e. of the manifest disease) does not mean a true etiological, microbiological healing or an elimination of the pathogen from the body. On the contrary, the parasite remains in

full vitality and full infectivity for other individuals, in certain organs of the body (e.g. the gall bladder), but the body no longer reacts to it as formerly with the typical symptoms (it may never have reacted); it has become tolerant. It is not the parasite which disappears from the body but only the symptoms of the disease; the cure is, as it were, only simulated. These symptomless, apparently cured, permanent excretors are of

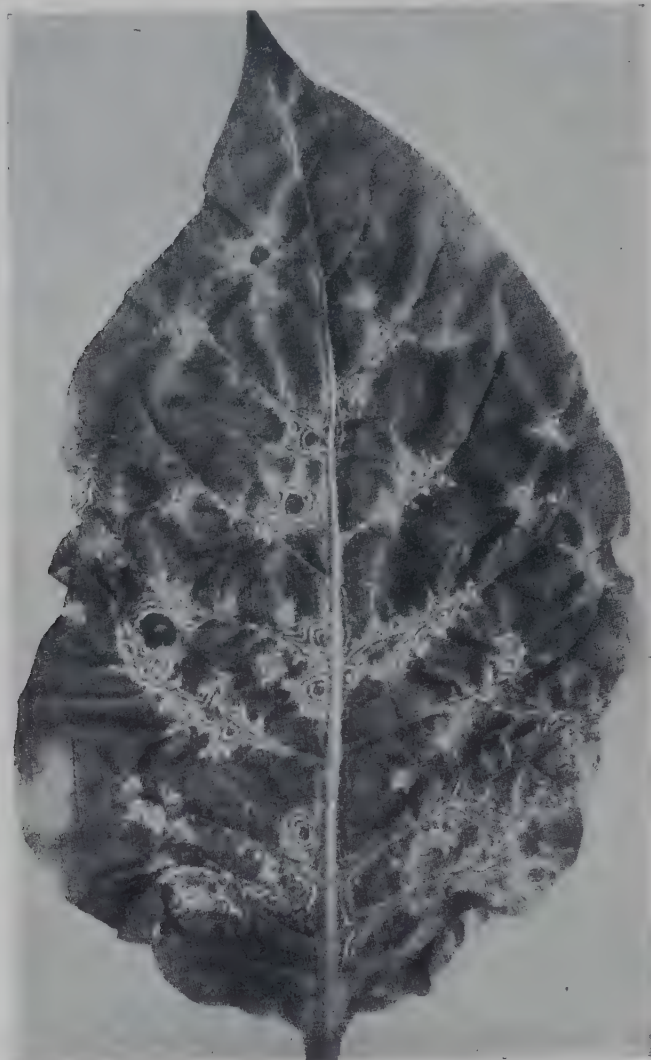


FIG. 223. Ring spot of tobacco (*Nicotiana tabacum*). $\times \frac{2}{3}$.
(After Wingard, 1928; from Gäumann, 1944.)

great importance epidemiologically since their acquired tolerance renders them symptomless carriers of the disease germ.

The 'cure' depends on two factors:

1. on true anti-infectious immune reactions which keep the residual infection in check and prevent it from breaking out again;
2. on readaptation of the body, a change in its reaction norm so that it tolerates the residual focus together with its metabolic products without showing symptoms.

The physician lays emphasis on the first group of these reactions because the major function of the human body is the localization of the pathogen

in a place where it cannot do any harm. If, finally, it tolerates the chronic residue of infection without manifest symptoms, there can be no further likelihood that the pathogen will regain its ascendancy. For this reason the physician classifies the typhoid example (and similar cases) as acquired anti-infectional immunity.

Botanical examples are somewhat different. In them, in contrast to typhoid, the anti-infectional defence reactions produce no such decisive result; in them the localization of the infection does not occur and the pathogen occupies the whole organism permanently but, nevertheless, is tolerated by it without manifest symptoms. Hence, the botanist emphasizes the second reaction complex, the desensitization of the body to the point of symptomless endurance of the pathogen which occupies all its tissues. The plant is thus more tolerant than man.

If, for example, the virus of the North American ring spot of tobacco be transmitted to fresh tobacco plants by rubbing the leaves, the typical symptoms (Fig. 223), concentric, pale, and partly necrotic rings which alternate with green tissue (Price, 1932, 1940) develop on the infected leaves after 3 days.

The virus spreads from the infected leaves into all the younger parts of the plants except the growing-point. Then the ring spot symptoms gradually begin to fade, those in the newly formed leaves and lateral branches become increasingly faint, and finally, after some weeks, no definite symptoms of the disease remain. The leaves are merely somewhat darker green, thicker, and more leathery than those of virus-free plants. Meanwhile, the originally diseased leaves have fallen off but the plants have 'preserved themselves'. They are clinically cured but remain virus carriers. The ring spot virus persists unweakened in all parts of the plant and is pathogenic to uninfected plants; only the symptoms have disappeared from the 'cured' individual.

The severe symptoms of primary infection (Fig. 224, *a*) are called the primary effect or primary lesions, and the mild symptoms of the following period the secondary effect (Fig. 224, *b*) or secondary lesions.

This clinical 'cure' depends on three factors:

1. On true anti-infectional defence reactions (production of virus antibodies) which ensure that the recovered leaves contain only about 10–20 % of the amount of virus in the primarily diseased leaves;
2. On a functional blockade of the remaining 10–20 % virus proteins. These are not inactivated since they are fully pathogenic to other plants, and it is only in the 'recovered' plant that they are unable to bring about the disintegration of chlorophyll or to cause the necrotic degeneration of certain tissues;
3. On the desensitization of the host organism, a conversion to tolerance, so that the host can tolerate the residual virus, although this assimilates for itself a considerable part of the host's native protein, without manifest symptoms.

The acquired tolerance can persist through at least ten generations of cuttings which remain symptomless virus carriers, but the tolerance is not transmitted through the seed. Further, if a healthy shoot be grafted on to the stock, the tolerance is not transmitted from the stock to the scion. The scion initially shows the symptoms of ring spot induced by the virus which has reached it from the stock and then, in its further growth, it repeats the processes leading to tolerance. The virus antibodies and the acquired tolerance are thus, in contrast to the virus itself, unable to migrate and circulate; only the virus migrates.

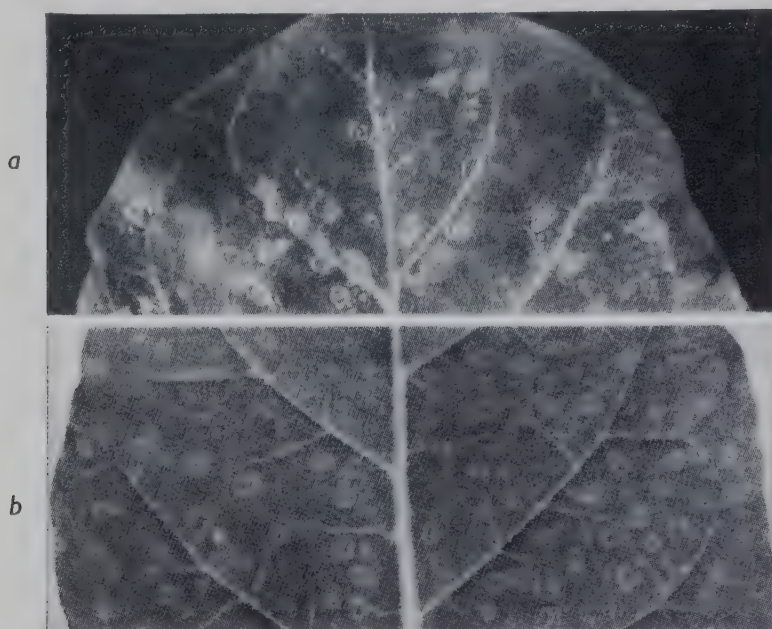


FIG. 224. Primary and secondary lesions on a tobacco plant, caused by potato virus X. *a* severe necrotic initial symptoms on the primarily infected leaf. *b* milder following symptoms on a later diseased leaf of the same plant. Nat. size. (After Köhler, 1940.)

If the clinically cured virus carrier be again heavily infected with the virus of ring spot it shows no symptoms; the recovered plants have thus become tolerant to a massive super-infection by the same virus. But, in this case, what happens is more than simple tolerance of the newly introduced virus; since the amount of virus in the leaves is not increased by a massive super-infection, something in the recovered leaves must counteract the increase of the virus. By means of appropriate experimental conditions it is even possible to bring about a symptomless primary infection so that the conversion may be effected without manifest prior disease.

The induced tolerance is group-specific for the ring spot viruses. Hence, plants remain susceptible to tobacco mosaic, &c., in short, probably to all viruses which infect tobacco but which are not related to ring spot mosaic. On the other hand, previously diseased plants are tolerant (i.e. are apparently resistant since they show no symptoms) to the virus of the yellow and green ring spot mosaic. Therefore, viruses developing in them are fully pathogenic for other plants; it is only the 'recovered' individuals that 'ignore' them and do not react to them.

The diagnostic features of 'induced' tolerance are, therefore, as follows:

1. A plant, during the course of an infection (or on account of a pre-infection still existing), undergoes an alteration of its reaction norm, which probably lasts only as long as the primary effect persists. Thus it appears to be an infection-bound tolerance just as premunity (p. 307) is an infection-bound immunity.
2. The continuing pre-infection, like premunity, is no protection against super-infection and (in contrast to premunity) also no protection against the generalization of the virus, but only a protection against the results of the new general infection.
3. The alteration of the reaction norm effects a desensitization, a neutralization, as it were, of the organism so that it no longer reacts to the general infection by producing disease symptoms; this contrasts with premunity which causes a sensitization of the individual (i.e. of its readiness for defence).

Nothing is known about the mechanism of these processes since the mechanism of virus action itself is still obscure.

In the example of the ring spot virus only the virus is transmitted by grafting, the induced tolerance does not pass from stock to scion. But in the case of certain other viruses, strangely enough, the tolerance accompanies the virus.

For example, if the virus of curly top of sugar beet be transmitted to young tobacco plants, some of them die and the remainder are stunted, developing short, crimped rosettes (like lettuces) instead of a main shoot. But new shoots may be formed from lateral buds and these contain the virus in full infectivity but show only mild disease symptoms. Like the parent plant they are tolerant of the virus and retain this toleration for at least four generations of cuttings. If these symptomless virus carriers be reinfected with the original virus from sugar beet (Fig. 225) their symptoms do not become more severe (Wallace, 1939); thus, they also tolerate a massive super-infection.

If scions from such 'recovered' plants are grafted on to healthy stocks, the latter become diseased but show only mild symptoms. They have undergone conversion to tolerance simultaneously with the virus infection. A similar result obtains if healthy scions are grafted on to a 'recovered' stock. A disease-abating effect is always exerted by a 'cured' partner. This also proves true if the originally healthy partner is massively super-infected with the virus.

Primarily infected plants can also exert this disease-abating effect but only if the disease has been present for a considerable time. Wallace (1940) infected groups of healthy tobacco plants with curly top virus at intervals of 5 days. On a given day the plants with 5-day-old infections showed no symptoms, the plants with 10- and 15-day-old infections showed moderately severe symptoms, and those which had been infected for 20 days a severe disease. Scions were taken from all four

groups and grafted on to healthy plants. The plants which received scions from plants with 5-day-old infections became severely diseased, those with scions of 10- and 15-day-old infections had symptoms of moderate severity, and those with scions from plants infected for 20 days exhibited only mild symptoms. Since it takes about 7 days for the union between the phloem bundles of the stock and scion at the graft to become established, it required nearly a month for the primarily infected tobacco plant to develop its disease-abating capacity to the full.



FIG. 225. Acquired tolerance of pre-diseased tobacco cuttings to the virus of curly top of sugar beet. Left: cuttings from none pre-diseased; right: from pre-diseased parent plants, infected anew with the same virus. Explanation in text. (After Wallace, 1939.)

Biologically, this transmission of induced tolerance is a remarkable process; the partner which has been 'cured' or which has suffered from the disease for a long time induces in the healthy partner a different reaction norm to the virus. It emits a specific antigenic stimulus, and the cells of the scion, which are affected by this stimulus, undergo a permanent modification and acquire the capacity to react to the effects of the virus less severely than before.

A similar distant action of one graft partner on the other is familiar in the organizer effect in animal transplantations (Spemann, 1936). Similarly, Haberlandt (1935, 1941) observed the same phenomenon in graft-hybrid or chimaeral plants of the *Crataegomespilus* type. *Crataegomespilus Asnieresii* is a periclinal chimaera with a skin one cell thick (epidermis) of medlar (*Mespilus germanica*) over a core of hawthorn (*Crataegus monogyna*). *Crataegomespilus Dardari* has two peripheral cell layers, an epidermis and a sub-epidermal layer of *Mespilus germanica*.

Sometimes reverted branches appear on these *Crataegomespilus* bushes. Thus, on *Crataegomespilus Asnieresii* they revert to *Crataegus* because the

Crataegus meristem at the growing-point breaks through the *Mespilus* epidermis and forms a genotypically pure branch of hawthorn (*Crataegus monogyna*). Conversely, twigs of *Crataegomespilus Dardari* revert back to *Mespilus* because only the two peripheral cell layers of the growing-point take part in the formation of new shoots. In this case, a genotypically pure twig of *Mespilus germanica* arises on a bush of *Crataegomespilus Dardari*.

Phenotypically, however, these reverted twigs are not true to either *Crataegus monogyna* or *Mespilus germanica*, they are intermediate between the two graft parents on which they occur. Thus, the stomata of *Mespilus germanica* average $32 \times 22 \mu$, of *Crataegus monogyna* $36 \times 32 \mu$, and of a *Crataegus* branch on *Crataegomespilus Asnieresii* $31 \times 24 \mu$, which is similar to those on *Crataegomespilus Asnieresii* itself. Also, the cuticular rings characteristic of *Crataegus* are found in stomata of the *Crataegus* twigs although their development is often irregular. On the other hand, the leaf hairs are, without exception, thick-walled as in *Mespilus*. Conversely, in the reverted *Mespilus* twigs on *Crataegomespilus Dardari* the *Crataegus* characters break through. Thus, in so far as periclinal chimaeras and not true hybrid grafts (burdos) are concerned, morphogenic substances from the deeper levels must be at work, which derive from the other partner of the chimaera and, as a distant effect, induce the phenotypic characters of a graft partner of a different species in the reverting twigs. The chemical nature of these morphogenic substances is still obscure, but the important point here is that, even in the plant kingdom, it is permissible to conceive of such a far-reaching induction effect of one graft partner on another.

A further example of such a long-range effect, in human medicine, is afforded by the immunity of adults to measles. The cells which form the body of the immune adult are no longer the same as those which came into contact with the measles virus in the body of the child because, in the interval, the body has renewed itself several times. Nevertheless, it has retained the capacity to defend itself immediately against a new infection of measles by energetic anti-infectional reactions and without recognizable symptom formation.

Two explanations of this phenomenon are possible; either the capacity of the organism for defence is continually being stimulated by constantly recurring latent infections or the capacity for heightened defence reaction is handed on vegetatively from one generation of cells to the next. Thus, not only the cells which were directly infected by the virus have the capacity for specific defence but also, on account of the long-range effect or permanent modification, their progeny.

Our knowledge of these long-range effects, as of so many pathological problems, has scarcely begun.

In virus research the phenomena of induced tolerance and premunity are utilized in determining the relationships of phytopathogenic viruses. Thus, the streak virus of tomatoes, in severe cases, produces striped or lined necroses on the leaves; hence the name of the disease. If tomatoes with streak disease be inoculated with aucuba mosaic, the latter virus

develops in the inoculated plants and can be demonstrated together with the streak virus in the distant young shoots, but it does not produce any symptoms, i.e. no yellow spotting (Caldwell, 1935). The streak disease has thus rendered the individuals tolerant to aucuba virus infection.

These mutual conversions to tolerance lead to the conclusion that the two viruses are related, i.e. that they belong to the same group; on the other hand, a negative result does not justify the opposite conclusion, i.e. that they are not related (Köhler, 1943). These methods may be supplemented by a serological technique resembling the ordinary medical precipitation methods, in which the plant sap is allowed to react with rabbit antiserum *in vitro* instead of *in vivo* (Spooner and Bawden, 1935; Stapp, 1943). But these serological tests also have their limitations. Thus, cucumber mosaic III and IV and tobacco mosaic should, on the grounds of their serological reaction, be related, but tobacco mosaic does not infect cucumber and cucumber mosaic does not infect tobacco (Ainsworth, 1935).

The practical question now arises: is it possible to use the phenomena of premunity and induced tolerance, by analogy with smallpox vaccination, for the practical control of disease; for example, could potatoes be saturated with an apathogenic virus in the field in order to immunize them against pathogenic viruses?

This can be done. For example, potato plants can be pre-infected with the apathogenic *H*-strain of the X-group of viruses, without showing disease symptoms. They also transmit the apathogenic virus to their vegetative progeny. If they are now super-infected with another virus of the X-group, e.g. the vigorous *N*-strain (closely akin to the *S*-strain mentioned on page 314, but which, in severe cases, produces an interveinal necrosis instead of a light green coloration) they do not become infected by it: they have become proof against all X-viruses (premunty). It was this protective effect that first drew attention to the symptomless *H* infection. If only X-viruses existed, then the control of potato viruses would be achieved.

Unfortunately the situation is not so simple. Firstly, the comparison with smallpox vaccination is only partially valid because the pre-disease artificially induced in our body is cured, the primary virus disappears, and it is the recovery process that induces the defence against reinfection. The plant virus diseases, on the other hand, are not cured, the attacked plants never become 'healthy' again but remain chronic virus carriers with all their associated latent dangers. Thus, the capacity of plants to resist frost may be lowered (as a physiological secondary effect of the symptomless infection) or external factors (e.g. temperatures above 35° C.) may cause the apathogenic virus to mutate and give rise to highly pathogenic strains.

A further difficulty arises, in that the symptomless disease of the plant protects it only against the X-viruses but not against the viruses of other groups, e.g. the malignant viruses of the Y-group and leaf roll. Also, the opposite effect may be produced; the super-infecting virus may, on occa-

sion, cause a more severe disease in the pre-infected individuals than it would by itself, either because the pre-infection increases the susceptibility of the hosts to these other viruses or, because of complex formation (activator effect, p. 228), the pathogenicity of the super-infecting virus is greatly increased.

To return to our example, the *H*-strain of the potato-X-virus group is also apathogenic to tobacco (causes no symptoms), and protects it against the other strains of virus within the X-group. The ordinary tobacco mosaic, which belongs to another group, is only slightly pathogenic to tobacco (Fig. 226, right) producing on the leaves only a faint interveinal mottle. But if this (in itself weakly pathogenic) tobacco mosaic be transferred to a plant carrying the *H*-strain, severe injuries, necroses, &c., are produced (Fig. 226, left). Thus, protection against the X-viruses would be purchased at the cost of an enhanced risk of disease due to other viruses. If smallpox vaccination rendered people much more susceptible to measles and poliomyelitis, it would scarcely be employed as a general prophylactic measure.

The extent of the practical danger to potato culture due to the formation of virus complexes cannot be finally assessed in the laboratory and must vary greatly in different areas of cultivation. Thus, it is clear that, under English conditions, the chronic Z infection (p. 110) has not acted as a limiting factor in the culture of the variety King Edward.

The idea of a prophylactic infection of potato plants in the field by apathogenic viruses undoubtedly deserves further investigation and it may possibly be achieved by means of polyvalent premunization; but much more work needs to be done before it can be recommended as a practical measure.

§ 2. Passive Immunization

In active immunization the antigen is introduced into the body to be protected and there stimulates the desired reactions, in particular, the formation of antibodies (p. 279); in passive immunization, however, it is not the antigen but the ready-made antibodies which are introduced (antibody



FIG. 226. The activator effect of apathogenic viruses. Left: a tobacco leaf pre-infected with the apathogenic *H*-strain of potato virus X, and super-infected with the mild common tobacco mosaic. Right: tobacco leaf inoculated with common tobacco mosaic only. (After Salaman, 1938; from Gäumann, 1944.)

injection in contrast to antigen injection). Thus, the body does not form the protective substances itself but receives them ready for use from another previously diseased or recovered body. If the incorporated antibody-containing serum comes from a different species (as in the control of acute diphtheria with antitoxic horse serum) the passive immunity is termed heterologous; if, on the other hand, the serum comes from the same species (as in the control of scarlet fever by serum from convalescents) it is termed homologous. In neither case is the body protected against the parasite, only against its toxins (saturation with them).

In plant pathology, passive immunization has been attempted with antibodies of both animal and plant origin. Among the experiments with the former, the work of Carbone and Arnaudi (1930) may be mentioned; they produced a rabbit serum highly agglutinative to *Bacterium tumefaciens* and this, applied to the cut surface of pelargonium shoots, gave complete protection against canker. Kaliaew (1935, cit. Savulescu, 1936) also observed the protective effect of rabbit antiserum against *B. tumefaciens* infection and also the healing effect on tumours already in existence. Thus, it appears that in certain cases a passive immunization of plants by animal antisera is possible.

On the other hand, experiments with homologous antibodies of plant origin are more contradictory, especially in the case of graft-scions and transplanted tissues. Does there exist in them a protective action which is based on mobile antibodies capable of circulation, and can this pass from a resistant or recovered stock to the scion and vice versa? It is known that glycosides, alkaloids, proteins, and viruses can effect this passage; it is also known that in curly top disease of beet (p. 347) the protective effect travels from the stock to the scion.

In the bacterioses and mycoses, however, evidence for passive immunization is mostly negative. Wormald and Grubb (1924) noticed that in apple trees a stock susceptible to *Bacterium tumefaciens* becomes resistant when grafted with a resistant scion and explained this by circulation of antibodies. On the other hand, in other diseases such as wart disease of potato (*Synchytrium endobioticum*; Roach, 1923, 1927) and in potato late blight (*Phytophthora infestans*; K. O. Müller, 1931) no passive immunization of the graft partner takes place. This obviously does not eliminate the possibility that protective substances are formed in the attacked cells, but only that these substances do not move out of the cells or do not move far enough, and also that they cannot be transferred in expressed sap.

§ 3. Defence Reactions and Immunity

From what has been said above it is clear that the plant cell, like the animal and human cell, possesses not only the usual vegetative functions of assimilation, growth, division, &c., but also the capacity to defend itself against disease agents (defence or immune reactions).

It is unnecessary to argue which of the two components (resistance or the capacity for defence reaction) is more important to the plant; they are

both vital to life. In comparison with human medicine, however, it may be said that problems of resistance still play a larger part in the study of plant infections than in those of animals and man; but, even in plant pathology, scientific interest is shifting more and more from pre-infectious resistance problems to those of post-infectious behaviour. The question most frequently asked now is not whether or why an individual becomes infected, but how it behaves during the course of its disease.

In so far as these reactions have a defensive character they conform in principle to those of the human body. All that the plant can do man can also do, but he can do still more. These differences may be compared under four main headings: (1) the mechanism of defence reactions, (2) their topological range, (3) their degree of efficiency (their utility), and (4) the duration of induced protection.

1. The mechanism of defence reactions. Plants and man have in common certain protoplasmic biochemical defence reactions (the formation of agglutinins, lysins, virus antibodies, &c.), and some histogenic demarcation reactions; in addition, however, in the anti-infectious group, man also possesses phagocytosis, and in the antitoxic group, the capacity to form specific antitoxins. These capacities are not possessed by plants to the same extent, but, on the other hand, plants have a greater capacity than man for aborting infection through local necrosis (necrogenous abortion).

2. The topological range of the defence reactions. The local cellular defence reactions are similar in plants and man, but man possesses in addition humoral immune reactions in which either the blood is responsible for the defence or the whole body is a sensitized substrate.

An experiment performed by Grumbach (1928) in following up work of Besredka (1921) will illustrate these local defence reactions of warm-blooded animals with which botanists may not be familiar. Grumbach injected the filtrate from an old culture of *Streptococcus* into marked places on the belly skin of guinea-pigs. After 48 hours he injected into the skin lethal doses of a highly virulent culture of the same *Streptococcus* at distances of 1, 2, 3, and 5 cm. from the original points of injection. Within an area of about 1 cm. from the point of injection an increased reactivity was produced by the previous treatment, so that the lethal dose was simply tolerated (Fig. 227). At a distance of 2 cm. only an inhibitory effect was noted which, however, was not sufficient to save the animal from death. At distances of 3 and 5 cm. from the point of injection there was no protective effect and the animals died.

The cells and tissues concerned have thus been sensitized by the intracutaneous vaccination with streptococcal filtrate and, as a result, they show an increased reaction potential whereby the invading micro-organisms are arrested close to the focus of infection and destroyed; only a local inflammation is produced. This heightened reaction potential is confined exclusively to the tissues in the immediate neighbourhood and it does not

include any antibodies freely moving in the blood; thus it does not result in an increased readiness for defence in the remaining parts of the body. The blood, therefore, is not responsible for these local defence reactions; they consist of true cellular protoplasmic reactions which extend only by infiltration from the reacting group of cells to the immediately adjacent tissues and to these only.

In addition to these local cellular defence reactions, man also possesses the familiar humoral immunity reactions which protect the whole body

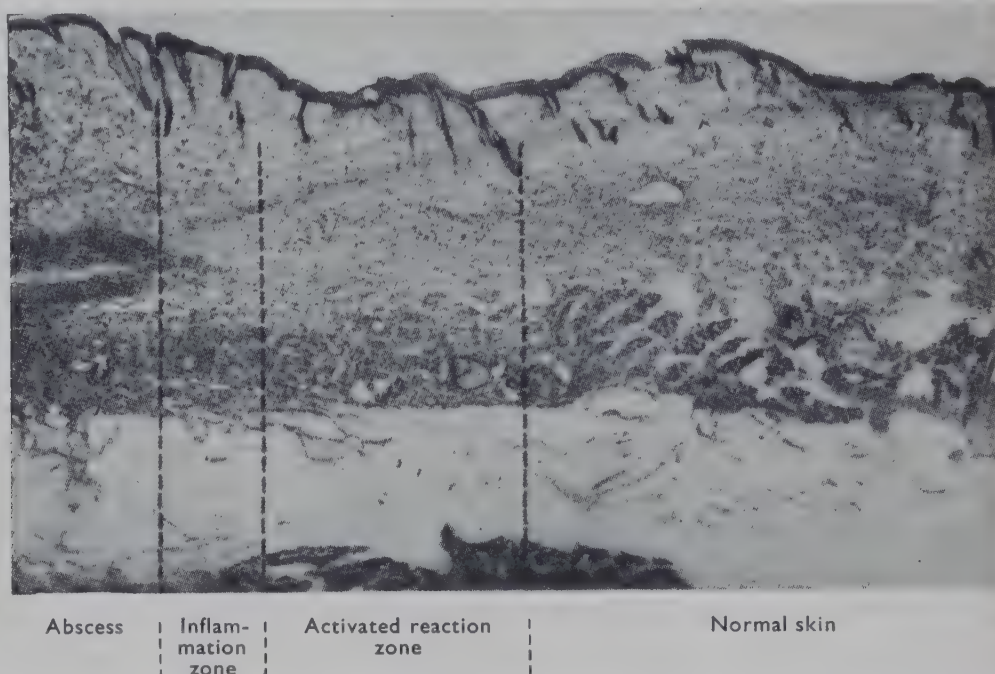


FIG. 227. Local streptococcal infection in the vaccinated abdominal skin of a guinea-pig. Explanation in text. $\times 40$. (After Grumbach, 1929.)

and generally obscure and overshadow the local reactions within a short time. On the other hand, the plant's defence reactions are characteristically local, they involve only the cells and tissues immediately affected and, therefore, do not lead to any general protection nor to a heightened defence readiness of the whole organism; i.e. they produce no immunity. Because of this confinement of the reaction potential to the focus of infection and its immediate neighbourhood, local defence reactions are common in plants which, however, show no humoral immunity.

A subsequent new infection on a distant part of the body usually develops independently of earlier infections elsewhere. It is, therefore, uncertain whether the plant's defence reactions (except those of the viroses discussed on p. 350) will ever be evaluated as prophylactic or therapeutic in the same way as the humoral reactions of animals and man.

This difference of grade between the topological range of the defence reactions of plants and those of animals and man is conditioned by the different organization of their bodies. On the one hand, outwardly, the plant is much more exposed than man (infection, p. 12) but, on the other hand, inwardly, it is much less of a whole than man; it has, as it were, never

grown up. The organization and coordination of a plant are at a lower level. Thus, if the top of a tree be cut off, water is still pumped up from below by the roots, either because they have not been 'informed' of the loss of the crown or because they cannot reduce their activity if they are 'informed'. Hence, we cannot expect from the plant body total reactions like the humoral immune reactions of the human body.

For the long-distance transport of materials the plant lacks a central circulatory system corresponding to the blood and lymphatic systems and a transport medium corresponding to the blood; instead of quick circulation in the plant body there is slow, ineffective diffusion along conduction gradients and from cell to cell. Thus, from the beginning, the humoral total immunization of the plant body meets with very great difficulties.

3. The effectiveness of defence reactions. Both the plant and man can weaken and localize the pathogen. But, in many infectious diseases, man can also eliminate the pathogenic agent and effect a cure, which the plant can seldom do. The level of immunity in plants is therefore low.

For example, human immunity reactions are 100% effective in those ordinary infectious diseases which, in favourable cases, end in a spontaneous healing, where the body recovers and eventually a regeneration of the destroyed tissues takes place. It is considerably less effective against syphilis, only curbing the spirochaetes but not eliminating them. Therefore, this disease, as a rule, does not heal spontaneously (i.e. without the chemo-therapeutic measures of the physician). Finally, it is almost completely ineffective in cases of massive tetanus and anthrax infection where, at most, the fatal outcome is only somewhat delayed.

The effectiveness of plant immunity reactions corresponds, in the main, only to the syphilis-tetanus level. It may also happen in plants that the focus of infection is eliminated by hyperergic reactions (as in the wart-immune potato varieties or the mistletoe-immune pear trees) and the lesion then really heals. But these cases are in a minority.

In plants the opposite is more usual. If the resistance factors and the first defence reactions fail and the infection 'takes', the lesion, as a rule, never heals but persists in the tissues once they have been attacked. In favourable cases, however, it is localized and generalization is thus delayed or prevented.

The lower efficiency of plant defence reactions is, perhaps, related on the one hand to a lesser antigenic effect of plant parasites, and on the other hand to a lower capacity for reaction of the plant body due to the special organization of its cell contents. Thus, animal cells are completely filled with protoplasm, whereas adult plant cells contain only a thin layer of protoplasm lining their walls whilst the remainder of their volume, perhaps nine-tenths, consists of vacuoles filled with a watery solution of salts. Only the protoplasmic layer can react to the parasite and, therefore, the part of the plant body which is capable of reacting is only a small fraction of the whole. For this reason, a reaction intensity comparable to that of animal and human reactions cannot be expected from the

plant body. The level of efficiency of plant defence reactions can only be low.

In the realm of plant life these limitations do not necessarily have fatal consequences. However, most plants do not live as long as man, they complete their lives in one or more years before a slowly progressive infection can become a danger to them. In addition they shed their leaves, needles, and even branches either every autumn or during the course of a few years, and if, in the meantime, these have become infected, they thus get rid of any local infections. Newly forming organs usually begin life in a healthy condition.

4. The duration of induced protection. The innate readiness for defence and the raising of its efficiency by sensitization are common to plants and man. In man, in certain cases, such as in 'children's diseases', this sensitization may last for life, in smallpox for about 10 years, in erysipelas (streptococcal infection of the deeper skin-layers) possibly a few weeks, whilst no measurable lasting protection against reinfection (at most a change in reaction norm) is produced by recovery from tetanus, tuberculosis, and certain skin diseases (*Microsporon*, favus).

Only the last type occurs in plants. In them the sensitization disappears along with the infection, since they have very little capacity for retention. As in tetanus and tuberculosis, their defence reactions serve only against an existing infection and do not act as a defence against a subsequent attack.

Because of this dependence on the presence of infection, infection-bound immunity (premunity) and infection-bound tolerance are the specific forms of plant defence against disease. In plants the readiness for defence can be increased but this heightened readiness lasts only as long as the stimulus (the storage of injection material presents a similar problem in diphtheria). The sensitization disappears with the stimulus since the plant is ahistoric. It is because of this limited capacity for retention that innate defence reactions are common in plants, but there is no acquired immunity.

For the kind of life that plants lead these inadequate defence reactions may suffice, but to the botanist they are regrettable. He must resign himself to the fact that although defence reactions occur in plants they are both quantitatively, in respect of their intensity, and qualitatively, in respect of their character and the duration of their effect, far behind those of the warm-blooded animals and man. Only a consideration of their phylogenetic development throughout the Metazoa (Rössle, 1923; Baer, 1944) would throw light on these problems.

III. THE INHERITANCE OF DISEASE PRONENESS

The innate infection- and disease-resistance capacities, as shown in the previous section, depend on a number of very heterogeneous conditions. Only seldom do they depend on a single factor but mostly they are the result of the interplay of heterogeneous elements such as resistance to attack, penetration, or spread, anti-infectional and antitoxic defence reactions, and induced tolerance. Accordingly their mode of inheritance is

very complex. Certain factors associated with resistance are inherited, as also are certain functional capacities associated with the defence reactions; the final result (the resistance to disease as measured experimentally in laboratory and field trials) is produced by the spatial and temporal co-operation of all these factors (Hansen, 1934; Roemer *et al.*, 1938).

The following facts are basic:

1. Resistance to any one disease is usually inherited as a unitary, genetic complex, entirely independent of resistance to any other disease.

2. For specialized pathogens (and these form the overwhelming majority) the resistance to each single strain is, as a rule, inherited as a unitary, monovalent, genetic complex, more or less independent of the resistance complexes against other strains of the pathogen. Thus, in any particular variety of wheat there is not one genetic complex, 'black rust resistance', but numerous complexes affording greater or lesser resistance to different strains of black rust. Only in comparatively few cases does the same resistance capacity afford protection against several strains of the pathogen (group resistance; polyvalent resistance complexes). For instance, Aamodt (1927) noted that, in one case, resistance to no less than twelve biotypes of black rust of wheat depended on a single factor; and Straib (1934) recorded a similar group-factor for nine biotypes of yellow rust of wheat.

In general, the more highly specialized a pathogen, the easier it is to breed varieties which are resistant to certain of its strains. Clover rot (*Sclerotinia ciborioides* = *S. trifoliorum*) is polyphagous and attacks species of *Medicago*, *Onobrychis*, &c., as well as *Trifolium* spp.; consequently no resistant varieties of clover are known and it will not be easy to breed one. On the other hand, with the highly specialized black rust of wheat the resistance is always effective only against a given biotype of the parasite and, therefore, wheat varieties with the appropriate limited resistance can be produced.

In regard to the number of genetic factors concerned, resistance complexes may be monohybrid, dihybrid, or polyhybrid. Monohybrid inheritance was observed by Schreiber (1932) in the resistance of beans to three strains of *Colletotrichum Lindemuthianum* (anthracnose); dihybrid inheritance by Mains *et al.* (1926), for the resistance of certain varieties of wheat to biotypes 5 and 12 of *Puccinia triticina* (brown rust); and polyhybrid inheritance by Seeliger (1925) and Kobel (1929) for the resistance of grape-vine to *Plasmopara*. On occasion, dominance, recessiveness, and crossing-over are also involved.

In other diseases the inheritance of resistance complexes does not follow simple Mendelian rules. Thus, potatoes are probably tetraploid and K. O. Müller (1930) assumed that their resistance to *Phytophthora* depended on four allelomorphic genes all of which must be present together in a given variety if effective resistance is to be produced.

Lastly, favourable mutations are also possible. Freisleben and Lein (1942), using Röntgen-rays on a spring barley (*Hordeum distichum*) susceptible to mildew, were able to produce individual plants which were

resistant to the three most important biotypes of mildew (*Erysiphe graminis hordei*) without showing any other recognizable constitutional change.

Occasionally resistance complexes are linked with others. These factor linkages can occur between the resistance complexes against different strains of the same pathogen, or between the resistance complexes against different pathogens, or between given resistance complexes, on the one hand, and given morphological or physiological peculiarities of the host plant on the other.

Factor linkage between the resistance complexes against different strains of the same pathogen was observed by Straib (1934) in a certain wheat cross resistant to the yellow rust biotypes 15 and 17. One of the primary aims of breeding combinations is to produce group-complexes resistant to the greatest possible number of biotypes irrespective of whether they depend on polyvalent complexes or factor linkages.

Weak linkages between the resistance to different pathogens has been noted in certain oat crosses, e.g. between their resistance to loose and covered smut (Reed, 1935), and in certain potato crosses, e.g. between wart- and *Phytophthora*-resistance (K. O. Müller, 1930). Naturally, such factor linkages are more desirable than those in which the resistance to one disease is linked with an increased susceptibility to another.

In those cases in which certain resistance complexes are linked with certain morphological or physiological characteristics of the host plant, causal connexions must be distinguished from true linkages. When a given disease resistance is due chiefly to certain factors of resistance to attack (e.g. *Claviceps* resistance in wheat and barley depending on the closing of the glumes, p. 257) or on certain factors of resistance to penetration (e.g. the thickness of the cuticle in black rust resistance of *Berberis* leaves; Table XXXVIII), then the relation between the disease resistance and the structural characters of the host is 'objectively' determined. In this case it is not possible to breed resistant varieties which do not possess the decisive characteristic.

In other cases there is true linkage, determined by the spatial location of the genes concerned in the same chromosome. In crosses between certain varieties of *Hordeum distichum* and *H. vulgare*, the resistance of the first parent against strains 1 and 4 of *Erysiphe graminis hordei* depends on a single factor which is linked weakly with the factors for hood-formation and number of ear rows (Freisleben and Metzger, 1941). In Svanhal barley three genes are concerned in resistance to stripe (*Helminthosporium gramineum*), each belonging to a different linkage group; gene 1 is linked with the factor for the two-row character, gene 2 with white colour of the glumes, and gene 3 with roughness of the grain (Griffée, 1925).

As a rule, when the capacity for disease resistance is inherited it has proved to be dominant. This can be explained by the spontaneous elimination of individuals with dominant disease susceptibility during the course of thousands of years. Yet, in breeding work, the likelihood of

obtaining complete success is very small owing to the great number of considerations which are necessarily involved in a breeding programme. For example, wheat must combine a good growth habit (demands from soil and climate, strength of straw, time of ripening) and a high technical quality (yield, baking quality) with resistance to the races of black rust, brown rust, yellow rust, covered and loose smuts, ear blights, foot rots, winter killing, ergot, stripe (*Helminthosporium*), &c., which are important in the relevant area of cultivation. The aims of potato breeding have been stated as follows by Roemer *et al.* (1938, p. 156):

'It is essential that every newly bred variety should be wart-immune, but it need not be immune to scab, nor initially need it be a good table potato, for that matter it need not be a table potato at all. It is more important that it should have a high starch content (fodder potato or industrial potato) because much the greater part of the potato harvest is used for these purposes. If it were possible to cultivate *Phytophthora*-immune varieties of non-edible potatoes then the fields with edible potato crops would also become less menaced by *Phytophthora*, because the extensive potato fields of the other varieties would no longer form sources of infection. Thus, it is obvious that the first *Phytophthora*-immune varieties need only possess the following characters: middle late to late maturity, high yield, abundant starch, wart-immunity and hardness. On the other hand, we should not, at first, demand from such *Phytophthora*-immune selections that they should have a particular colour of flesh, skin or tuber, depth of eye, and resistance to scab. These are demands which we can make and fulfil later.'

In addition to these very great objective difficulties, it must also be borne in mind that the pathogen itself is constantly producing new strains. A host variety resistant to a given strain of a pathogen does not remain resistant for ever. Wind and world trade are always introducing new strains, against which the artificially bred resistance-complex is no longer effective. New strains of the pathogen arise spontaneously through hybridization, mutation, &c. The outlook of the plant breeder is distinctly pessimistic; he can never reach his goal for this always seems to move farther away, but he is satisfied if he can keep ahead of the parasite for anything up to 10 years.

B. CHANGES IN DISEASE PRONENESS DUE TO THE ENVIRONMENT (THE DISPOSITION OF THE HOST)

The fluctuations in intensity shown by a given infectious disease of plants in different regions and years was, for a long time, explained by a selective favouring of the development of the pathogen; e.g. that the developmental conditions in warm, damp regions or summers were more favourable than those in hot dry ones. Only later was it realized that the environmental factors not only influence the growth and form of the host plant but also, on occasion, its disease proneness. The environment not only provides the conditions necessary for infection (p. 19) and not only

alters the parasitic disposition (the aggressiveness) of the pathogen (p. 192) but, under certain conditions, it also modifies the disease proneness, i.e. the susceptibility and resistance of the host.

The constitutional susceptibility and resistance mentioned in the previous section is, in most cases, not a fixed, constant value but merely a range, an hereditarily determined zone of fluctuation or a genetically fixed region of variation, in which the actual infection- and disease-proneness of a given individual can shift reversibly to the positive or negative side. The current degree of proneness of a given individual to a given disease is termed its disposition; the plant is variably disposed towards the given infection.

Human pathology sometimes uses the expression 'disposition' in a wider sense as a synonym for susceptibility and speaks of an innate disposition and an acquired disposition. In plant pathology a narrower meaning is given to the term. Disposition is a phenotypic, temporary, reversible state of susceptibility within the limits of the innate genotypic variation range; the innate disposition is termed the constitutional susceptibility.

Thus, in individuals with such labile disease proneness, the disease is 'caused' not merely by the pathogen but also by the immediate condition of the individual. Sometimes the fact that disease proneness can itself be an expression of an internal disturbance of the organism is a sign that the internal adaptation can no longer cope with the outside influences (disturbance of the internal equilibrium).

In such cases, the environmental conditions favour disease to such an extent that the pathogen may be said merely to initiate it. The pathogen in this case is not the sole cause but the *conditio sine qua non* of the disease.

A cursory glance at civil lawsuits shows the importance of these problems. Industrial fumes can increase the disease disposition of certain crops. If the parasite be not present in the given region, then the heightened disposition will not become manifest, but, if the disease be already present, its development will be favoured by the external conditions. How much responsibility for the damage is attributable to the plaintiff and how much to the defendant? There is here great opportunity for biological controversy.

The range or scope of the innate zone of variation of disease proneness differs according to the species of host and pathogen but, to a certain extent, it is characteristic for a given disease. Completely susceptible plants become diseased whatever the environmental conditions, whereas completely resistant plants never become diseased; neither show any 'disposition'. It is primarily in types with intermediate disease proneness that the environmental conditions can effect modifications.

As a rule, the labile, plastic variation zone is about as great as the range of the genetic susceptibility that can be affected by breeding. In forms whose susceptibility can only be slightly reduced dispositionally, the prospects of successful breeding are usually very slight.

Like the zone of variation, the lability, the degree to which disease proneness can be influenced, varies from one disease to another and is

again, to some extent, characteristic for each. For example, the susceptibility of rye to ergot can be altered only with difficulty. External factors can decrease only very slightly the 100% susceptibility of a given variety of rye; but, on the other hand, as will be seen in the course of this section, the rust susceptibility of rye may be affected by quite minor variations in external conditions (nutrition, temperature, light, &c.).

The 'disposition', which can vary, is a complex of factors. In the first place a distinction must be made between the influence of external factors on the establishment of the parasitic relationship (i.e. the disposition to infection) and the influence of external factors on the subsequent course of the parasitic relationship (i.e. the disposition to disease).

By the term 'infection disposition' is understood the proneness of the host organism to allow the disease germs to enter and establish themselves.

By the term 'disease disposition' is understood the proneness of the host organism to react to the stimulus emitted by the disease germs or by the injured cells.

TABLE XLVIII

*The influence of nitrogen nutrition on the infection- and disease-disposition of tomatoes to tomato wilt (*Fusarium lycopersici*). (After Cook, 1937)*

Tomato variety	Infection disposition (% of individuals infected)		Disease disposition (% of individuals diseased)	
	Low N	High N	Low N	High N
Bonnie Best (susceptible) . . .	45	67	45	56
Marglobe (resistant) . . .	10	70	10	0

It is not always easy to distinguish between infection disposition and disease disposition, but they need not coincide. In diphtheria, the number of symptomless infections is about thirty times as high as the number of actual diphtheria cases; the infection disposition is thus about thirty times as great as the disease disposition. Similar relationships occur in infectious diseases of plants. Table XLVIII shows two tomato varieties, one susceptible and one resistant to wilt disease, which were grown in sand culture at constant soil temperatures and with similar nutrients, except for differing amounts of nitrogen, and which were inoculated with conidia of *Fusarium lycopersici*. With abundant nitrogen, about 70% of the individuals, both of the susceptible and resistant varieties, proved to be spontaneously infected; in this instance the infection disposition of the tomatoes was increased by addition of nitrogen. The opposite holds true in the case of the disease disposition; in the resistant variety Marglobe with abundant nitrogen all infections abort, and the disease symptoms do not appear. The disease disposition of the tomato to 'wilt' is thus an independent behaviour complex and is influenced by variations in the nitrogen supply in exactly the opposite way to the infection disposition.

How greatly the disease disposition can vary from the infection disposition is shown by those cases in which, by reason of an infection disposition, permanent latent infection exists but, in the absence of disease proneness, cannot break out and cause disease (absence of disease disposition with presence of infection disposition). Certain pathogens causing wet rot of potato tubers, e.g. *Bacillus vulgatus* and *Bacillus subtilis*, can live for months in the vascular bundles of the tubers without giving rise to symptoms; only if there is a disturbance of the inner constitution of the host by high environmental temperatures (30 or 23° C.) do they become actively pathogenic (van Hall, 1902).

However, the number of infectious diseases of plants which can give rise to such latent internal infections is small, and it must be much more common for plants to be infected anew from the outside. Disparity between infection disposition and disease disposition is more important in human medicine. In such common infectious diseases as pneumonia, typhoid, and tuberculosis the disease germs can live for a long time as more or less harmless endophytes of certain parts of the body and they only become dangerous on the appearance of a disease disposition, i.e. if the body loses either its readiness for defence or its tolerance.

Thus, in general, the disease proneness of plants is less markedly affected by external influences than is the case with man. Hence, the pathogen dominates plant pathological thought, whilst human medicine is much more concerned with the immediate state of the host organism, which controls the interplay of forces between pathogen and host, and hence determines the clinical picture and the course of the disease.

Even with this splitting of 'disposition' into infection and disease moieties, the practical difficulties of this conception are not removed, since the content of both 'infection disposition' and 'disease disposition' is again very heterogeneous. Within the 'infection disposition' both the factors of resistance to attack and resistance to penetration (pp. 258 et seq.) and also the possibility of defence reactions (pp. 278 et seq.) may vary, and even, on occasion, in opposite directions under the same external influences.

Within the 'disease disposition' the following three factors may vary: (1) the fitness of the organism to serve as host; (2) its 'inclination' to do so (its capacity for defence, its level of immunity); (3) its reaction attitude, i.e. its 'inclination' to react to the infection pathologically. These three groups cover the true disease proneness which manifests itself, for example, in enfeeblement or in hyper-sensitivity to the pathogen (acute disease symptoms).

On account of the youthfulness and vast scope of plant pathology, there have been only few attempts to make a fundamental analysis of the factors involved in the above described situation.

So far as the individual factors are concerned, which can affect the disposition of the host, the first problem is to distinguish between internal causes and external influences.

The former effect those changes in disposition which necessarily appear during the course of the individual life, e.g. youth susceptibility and age resistance. On account of their fateful internal conditioning this disposition group (our sub-section 1) might equally well be termed constitutional resistance; it is merely a matter of opinion whether one says that the 'constitutional resistance' or the 'disposition' of an individual alters in the course of ontogenetical development.

External influences comprise all those factors of soil, climate, weather, &c., which, together, constitute the locality and the environment (Braun, 1937). Research is mainly concerned with two groups of problems: (1) the analysis of single factors and their range of action, i.e. the isolation and definition of the individual factors, and (2) the assessment of their importance, since this varies with the factor constellation; thus the influence of the environmental temperature or of the amount of light varies according to the amount of nitrogen supplied. The isolation of single operative factors has already made considerable progress, but their combined action (their mutual interference) and their varying importance from case to case still needs more thorough investigation.

1. *Ontogenetic Changes in the Disease Proneness of the Host*

The problem of ontogenetically conditioned change in disposition is not quite the same in botany as in human and veterinary medicine. The human being is young once only and grows old only once. His limbs and organs are all formed at the same time, and his body, despite the repeated renewal of its cells, usually persists through all the stages of childhood, youth, maturity, and old age.

On the other hand, the plant only partially conforms to this rule, since it is not a unitary organism. It has, indeed, definite, often characteristic age limits; a tree reaches only a certain age, then becomes decrepit, and finally disappears. Nevertheless, the foliage of an ageing tree is re-born every year and matures anew in every growing season. Moreover, the vegetation points continue to grow from the time the buds open until late in the summer and, therefore, at any given time the shoots have mature leaves at the base and young leaves at the tip. On one and the same individual, parts and organs in the embryonic state exist in harmony side by side with others that are in a state of senescent disintegration.

Hence, the plant organism undergoes two ageing processes, that of the individual as a whole and that of its single organs and parts (twigs, leaves, roots, fruits, &c.). These two processes are in a certain sense antagonistic, since the leaves arising from an ageing growing-point are the youngest. Life diminishes, therefore, with distance from the growing-point which is always producing new leaves, whilst the earlier leaves die (Paeck, 1940; Smirnov, 1928).

Fig. 228 illustrates the effects due to advancing age. Plants of the Indian wheat variety Pusa 4 were grown at a constant air temperature of 27.5–28° C., constant lighting, and constant carbon dioxide content of the air

(0.33–0.38%). At given intervals from the start of germination (abscissae) the amount of carbon dioxide in mg. per 100 sq. cm. of leaf-surface assimilated per hour was determined (ordinates) and, each time, values were obtained from young leaves (curve 1), mature leaves (curve 2), and old leaves (curve 3). After 70 days the plants began to flower; after 84 days the leaves began to yellow, and after 112 days to wither.

Two processes of ageing are superimposed in Fig. 228: (1) the absolute age, the age of the individual, shown along the abscissae; (2) the relative age, the stage of development of the single leaves, shown in curves 1–3.

1. The influence of the age of the individual. Under constant external conditions, the intensity of assimilation of leaves of equal age gives a curve

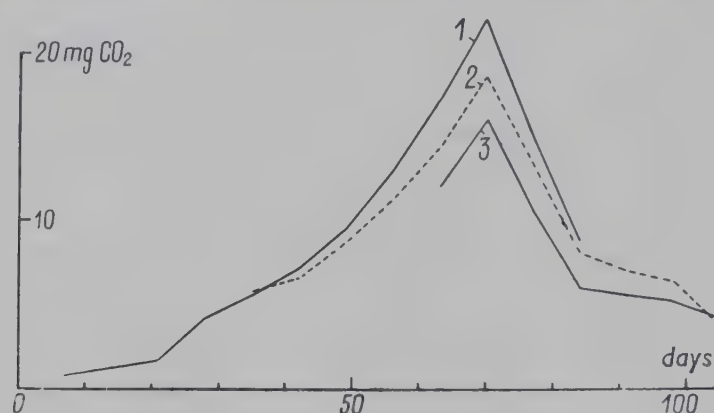


FIG. 228. Assimilation rate of young wheat leaves (curve 1), adult leaves (curve 2), and old leaves (curve 3). Abscissae: age of plants. Ordinates: assimilation rate. Explanation in text. (After Singh and Lal, 1935.)

with an optimum peak at the time of flowering. Young leaves of old plants assimilate less intensively than young leaves of young plants. The meristem, i.e. the growing-point, also grows old. The young leaves of a given individual do not all arise under the same conditions in the course of a growing-period.

2. The influence of the developmental stage of the leaf. The intensity of assimilation diminishes with increasing maturity; *ceteris paribus* old leaves (curve 3) assimilate less intensively than young ones (curve 1).

Such superimposed curves can also be obtained for the protein and carbohydrate status, &c. It can hardly be doubted, therefore, that these physiological changes can sometimes also lead to variations in disease proneness.

The differential susceptibility of various ages and developmental stages will be called phase disposition. Because two ageing processes overlap, special precautions must be taken in cases (e.g. cereal rusts) in which the individual disposition of single organs has to be watched with particular care; thus, comparison should only be made (1) between leaves of the same age (i.e. leaves in the same stage of growth), and (2) between leaves of the same level of insertion (i.e. leaves originating at the same time in the life of the individual concerned).

Since plant individuals bear both old and young parts and tissues side

by side during the entire vegetative period, the problem of the ontogenetically determined change of disposition plays an incomparably greater role in plant pathology than it does in human and veterinary medicine.

But examples are also to be found in the latter. Infants do not develop lobar pneumonia. Blackleg or symptomatic anthrax is a specific 'children's disease' of cattle and 92% of cases are found in animals of 3 years old or less. On the other hand, in the closely allied para-blackleg, no such phase disposition exists. The *Ankylostoma duodenale* of man attacks only puppies and not grown dogs. Certain spirochaetes and trypanosomes attack poultry only in the egg and not after hatching (Doerr, 1941). Nevertheless, these are exceptions; the ordinary 'children's diseases' of man (measles, scarlet fever, whooping-cough) do not depend on a youth disposition (i.e. are not phase specific children's diseases) and occur regularly in us only in childhood because, owing to social conditions, we are infected early, fall ill, and subsequently acquire immunity; in regions where these diseases are not endemic adults are also infected. Conversely, the age disposition in man manifests itself more in non-infectious diseases, for instance, in a proneness to cancer and functional disorders of the heart and blood-vessels.

According to the place at which the ontogenetically determined change of disposition measurably affects the resistance system of the plant, alterations can be distinguished in (a) resistance to attack, (b) resistance to penetration, (c) resistance to spread, and (d) level of immunity.

(a) *Ontogenetic Changes in Resistance to Attack*

In the ear blights (fusarioses) of cereals there is a close inter-relationship between the stage of development of the ears and the incidence of disease, since the vital organs are accessible only at a very early age. Early infections during the youth of the grain (between flowering-time and early ripening) attack the primordium of the coleoptile and particularly the scar (the hilum, the place of attachment of the funicle). Consequently, the entry of nutrients through the hilum is obstructed, and the attacked grains develop poorly (shrivelled ears, Fig. 229) and show a marked decrease in their germinability. In late infections, between yellowing and full ripening, the germ tubes of the conidia germinating on the grain can penetrate only into the seed coat, the grain develops normally, no infection is visible, and a decrease in germinability cannot be established.

Other good examples of ontogenetic changes in resistance to attack are given on pp. 253 et seq.

(b) *Ontogenetic Changes in Resistance to Penetration*

Ontogenetic changes in resistance to penetration are extraordinarily common in plants. Many plant parasites are little if at all able to penetrate outer layers of cutin, cork, &c., or mature cells with cellulose or lignified walls and are therefore dependent on young meristematic tissue; accordingly, the ontogenetic changes in resistance to penetration in plants mostly

tend to pass from susceptibility in youth to resistance in age. Further, since the maturing of the peripheral layers often occurs simultaneously with that of the ground tissues, the ontogenetic heightening of resistance to penetration usually runs parallel with that of resistance to spread, and it is hard to differentiate between the two.

Four organ or tissue groups are especially involved in ontogenetic increase of resistance to penetration: seedling tissue, leaves, cortex, and fruit.

In the seedling the very high susceptibility to disease of young or meristematic tissue results in special seedling diseases. In sugar beet, spinach, cress, forest trees, &c., they are caused by *Pythium de Baryanum*,

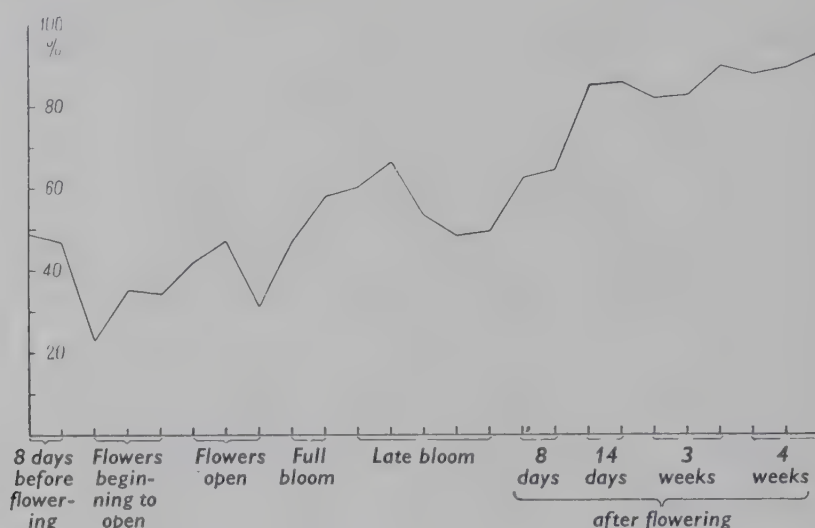


FIG. 229. The influence of ageing of rye ears on their susceptibility to ear fusariosis (*Fusarium culmorum*). Abscissae: developmental stage of the ear. Ordinates: grain initials as % of flower count. (After Baltzer, 1930.)

&c. ('root rot'); in beech, maple, &c., by *Phytophthora fagi*; in cereals (e.g. snow mould) by *Fusarium culmorum*, *Gibberella Saubinetii*, &c. They appear predominantly in the seedlings, because here the embryonic tissue and, usually, external conditions favourable to infection are at their disposal. They do not, however, constitute true phase-specific 'children's diseases' in the sense of the previously mentioned specific youth susceptibility of cattle, dogs, and poultry, since, under favourable external conditions, young tissues of adult individuals are also attacked. Thus, *Pythium palmivorum* attacks the 'heart' (the young, still folded leaves) of the coco-nut palm, &c.; *Phytophthora cinnamomi*, the young bark of the cinnamon tree; and the Fusaria of cereals mentioned above also attack the roots (foot rot) and the culm and young ears (ear blights, shrivelled grain) of mature plants.

The same is true for those fungi whose infection 'takes' with greatest certainty in seedlings (e.g. on the cotyledons, whence the mycelium penetrates into the young shoots) but also 'takes', although with less certainty, on other young tissues, e.g. in certain *Peronospora* spp. (p. 37) and cereal smuts (p. 50).

In leaf diseases those pathogens whose germ tubes penetrate directly through the cuticle and the outer walls of the epidermis in the manner

of *Botrytis*, are generally excluded by the increased thickening and hardening of the cuticle. For this reason, only young *Berberis* leaves can be used for infection experiments with black rust of cereals (p. 250), and only young peach leaves for infection with the pathogen of leaf curl (p. 93).

A similar ontogenetic increase in resistance to penetration also occurs in some leaf diseases in which the pathogen uses mainly the stomata; thus, according to Müller and Sleumer (1934), on a vine-stock the uppermost youngest leaves measuring about 20–25 sq. cm. are most easily infected with *Plasmopara viticola*; the lower the leaves are situated on the stock the more difficult it is to produce infection. In addition to the ontogenetic increase of resistance to penetration (e.g. due to variation in the diameter of the stomatal opening) an ontogenetic rise in the general level of immunity of the leaves also plays a part here. On the young leaves the conidiophores of the downy mildew are formed even at the comparatively low relative humidity of about 70–85%, whereas, on older leaves, they are formed only under optimal external conditions, viz. at a humidity of 80–100%. Such comparative observations, naturally, can only be made on leaves of the same stock and, if one wished to make use of one and the same leaf for the whole growing-period, then the ontogenetic decrease of susceptibility would be obscured by the favourable influence of mid-summer climatic conditions on the growth of the fungus.

An example of an ontogenetic increase in resistance to penetration of cortical tissues is afforded by the witches' broom of silver fir, caused by *Melampsorella caryophyllacearum*. Primary infection takes place, contrary to many statements in the literature, not in the buds, which later will become deformed, but in the young bark immediately around them. In the spring the germ tubes of the basidiospores grow into the epidermis of the unfolding shoot, then until the autumn the fungus grows a little farther into the cortex and produces a slight swelling of the shoot axis which, in the course of the year, may develop into a thick cankerous growth. If the infected zone contains buds then, in the following spring, these develop to produce the characteristic deformation. Instead of growing out horizontally like normal shoots, they grow vertically and the needles are arranged radially as in the main shoot of the silver fir, instead of pectinately. In the following year they branch and, since the branches are again not horizontal but more or less erect, they give rise to a bushy formation like a witches' broom (Fig. 230) that may continue to exist for up to 60 years.

The period of development during the elongation of the twig in which an ontogenetically determined susceptibility to penetration by the germ tubes of *Melampsorella* exists is thus short, and the probability that the basidiospore of the rust fungus will gain access is correspondingly slight. If one considers how minute is the amount of susceptible bark of young shoots in a forest, it is surprising that witches' brooms are so frequent.

Examples of fruits whose resistance to penetration (in contrast to resistance to spread, to be considered later) increases with age are very

common. Only the young grapes of the vine can be directly infected by zoospores of *Plasmopara viticola*. Later, the grapes come to possess an almost complete age resistance to penetration; the fungus can grow into them only from the stalk and causes them to shrivel like a tobacco-pouch: 'leather-berries'.

In the attack on tomato by *Macrosporium tomato* (leaf spot disease and fruit rot) the ontogenetically conditioned increase in resistance to penetration has been examined quantitatively. Young tomato fruits have a soft



FIG. 230. A witches' broom, about 7 years old, on white fir, caused by *Melampsorella caryophyllacearum*. (Orig.; $\times \frac{1}{10}$.)

skin and, in particular, a thin cuticle. As they ripen, the thickness and the mechanical strength of the cuticle increase and, parallel with this, there is an increase in the amount of pressure which must be exerted in order to penetrate it with a glass needle of 78μ diameter. This pressure increases (see Fig. 231) from 2.7 g. (in young fruits with an average weight of 7 g.) to 5.9 g. in fully grown fruits (average weight 254 g.), and thus it is more than doubled. If the fruits are sprayed with a spore suspension of *Macrosporium tomato*, up to 100% of the youngest fruits become infected. With increasing age and growing perforation-resistance of the cuticle, the incidence of attack falls, and reaches zero with a perforation-resistance to the 78μ needle of 5.5 g.: the cuticle can no longer be penetrated by the germ tubes.

Thus, during the course of their development, tomatoes acquire a marked resistance to penetration, which changes (in absence of injury) from complete youth susceptibility to a similarly complete age resistance (Fig. 232). The inside, the flesh of the fruit, remains, however, equally susceptible to *Macrosporium* throughout (like grapes to *Plasmopara*); hence, the low

resistance to the spread of the parasites that have penetrated remains unchanged. The practical difference depends only on the fact that the fruits, in the course of their life, acquire a 'thick skin' and, therefore, can exclude the parasites. In their cutin mantle they are protected like gold in a safe.

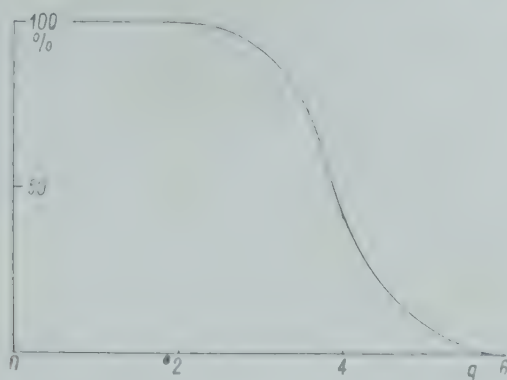


FIG. 231. Relation between the resistance to perforation of the cuticle of tomato and the susceptibility of the fruit to *Macrosporium tomato*. Abscissae: perforation resistance. Ordinates: disease incidence. (After Rosenbaum and Sando, 1920.)

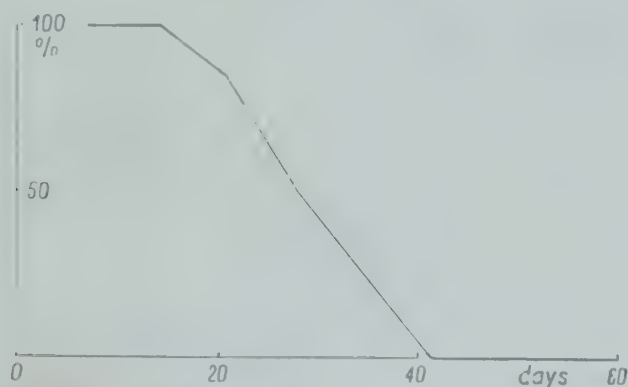


FIG. 232. Relation between the age of tomatoes and their susceptibility to *Macrosporium tomato*. Abscissae: age of fruits in days. Ordinates: % diseased fruit. (After Rosenbaum and Sando, 1920.)

(c) Ontogenetic Changes in Resistance to Spread

Resistance to spread can change ontogenetically either in a positive or in a negative direction.

A developmental increase of resistance to spread occurs particularly in those resistances which are associated with the structure of the cell walls; the strengthening, hardening, or even lignifying of the deeper-lying cell walls can increase to such an extent that every single adult cell is protected against numerous parasites by its wall alone (p. 263). Because of the fact that a serious hindrance to the development of many plant parasites already exists in the mechanical supporting structures of the plant body, passive resistance to spread plays an incomparably greater role in plant pathological considerations than it does in medicine.

Against *Ustilago zaeae* (maize smut; Fig. 144) the mature ground tissues show not only an age resistance to penetration but an equivalent age

resistance to spread. Hence, infection, whether through the epidermis or through wounds, is restricted to the small area in which meristematic cells are available.

Ontogenetical increase in resistance to spread is particularly important in wood. Under constant conditions of temperature and moisture and taking an average of twelve fellings spread over a year, mature wood of spruce (*Picea excelsa*) and fir (*Abies pectinata*) is only half as quickly rotted as young wood of the same trunks by the three dry-rotting fungi (*Merulius lacrymans*, *Polyporus vaporarius*, and *Coniophora cerebella*) or by the agent of shell-break (*Lenzites abietina*) (Table XLIX). Mature wood undoubtedly contains somewhat less nutrient material than young wood.

TABLE XLIX

The average amount of rotting in newly felled spruce and fir wood kept under constant laboratory conditions for 3 months. (After Gäumann, 1930)

Type of wood	<i>Merulius lacrymans</i>	<i>Polyporus vaporarius</i>	<i>Coniophora cerebella</i>	<i>Lenzites abietina</i>
Spruce:	%	%	%	%
young wood . .	40.3	36.6	23.8	45.6
mature wood . .	25.7	25.8	14.5	29.8
Fir:				
young wood . .	27.4	33.6	17.3	47.4
mature wood . .	18.4	18.8	11.5	25.4

TABLE L

The average amount of rotting of May-felled spruce and pine wood caused by *Merulius lacrymans* and *Polyporus vaporarius*. (After Gäumann, 1930)

Type of wood	Merulius lacrymans Decay after 6 months			Polyporus vaporarius Decay after 6 months		
	Green wood	Wood stored dry for one year	Increase in resistance to decay	Green wood	Wood stored dry for one year	Increase in resistance to decay
Spruce:	%	%	%	%	%	%
young wood	61.6	31.9	48.2	48.1	31.8	33.9
mature wood	47.2	24.6	47.9	39.2	24.2	38.3
Fir:						
young wood	56.0	26.7	52.3	53.1	24.0	54.4
mature wood	39.3	12.6	67.9	35.1	15.9	54.7

In this experiment (Gäumann, 1928 b) the total available carbohydrate (sugar, starch, hemicelluloses) in mature spruce wood averages only about 9.9% (against 13.1% in young wood), and in mature fir wood only about 8.3% (against 13.2% in young wood). The carbohydrate content of 9.9% or 8.3% would, in itself, be quite sufficient to allow the given fungi to grow abundantly. But, as enzymatic breakdown in mature wood is so much

slower than in young wood, this must be connected with the fact that the cellulose tissues become thicker and irreversibly shrunken with increasing age and, in consequence, the fungal enzymes are presented with a smaller area of attack.

It is this same process that renders felled spruce and fir more resistant to decay in storage than in their green state. In the experiments from which Table I. was compiled the parallel trunks to the green test trunks were stored dry for a year in an airy wooden shed, then sawn up and exposed to the fungi under constant optimal conditions of temperature and moisture. A loss of nutrients did not take place during storage, but the structure of the cell walls underwent a maturation, a hardening, an irreversible shrinkage (similar to hysteresis) with the result that its resistance to fungi increased from about 35% to about 60%. The practice of allowing wood to season for at least a year before using it for building is one of the reasons for the greater lasting quality of the wooden buildings of our ancestors.

In mature wood of certain kinds of tree (Scots pine, larch, oak, &c.) the ontogenetic resistance to spread is still further increased with the passage of years by the formation of heart-wood which stores tannin compounds, &c.; hence, the mature wood is still better protected from direct attack by the ordinary wood-destroyers and remains accessible only to the true heart-wood specialists.

In many fruits (berries, stone- and pome-fruits, &c.) the ontogenetic changes in resistance to spread are in the opposite direction, i.e. from youth resistance to age susceptibility, particularly in those infectious diseases which are generally classed together as 'rots'. Especially between ripening (i.e. the time between attaining maximum volume and edible ripeness) and ageing, the resistance to spread of the fruit flesh to the fungi concerned often diminishes materially. But it is difficult to establish the causes of these changes. It has been suggested, for instance, that in apples a direct relationship may obtain between their resistance to spread against the pathogens of storage rots (e.g. Fig. 233) and the developmental changes in their acidity, or in the dry weights of their sugar, nitrogen, tannin, pectin, raw fibre, and ash content (e.g. Figs. 234 and 235). But, contrary to expectation, the various analyses have not led to any unequivocal conclusions.

There is certainly a relationship between the sugar and acid content of

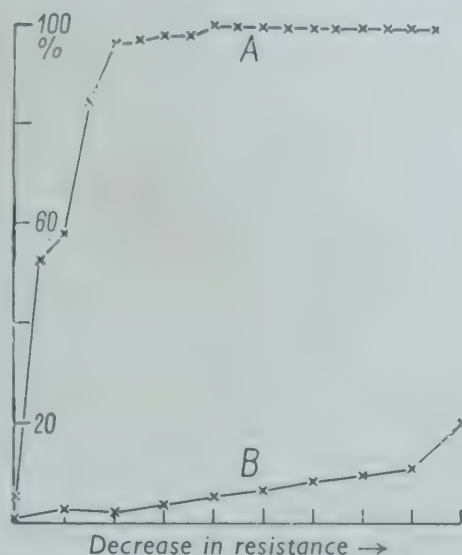


FIG. 233. The rate of spread of a strain of *Botrytis* (curve A) and a strain of *Fusarium* (curve B) in apples (var. Cox's Orange Pippin) at different stages of maturity at a storage temperature of 12° C. Abscissae: decrease in resistance to spread. Ordinates: % rotten tissue. (After Gregory and Horne, 1927.)

the substrate and the growth of the fungus, but it is not a two-dimensional one because the acid action varies with the sugar content (Fig. 236) and the action of the sugar with the acid content (Fig. 237). Similar complex reciprocal actions exist between the nitrogen and acid content

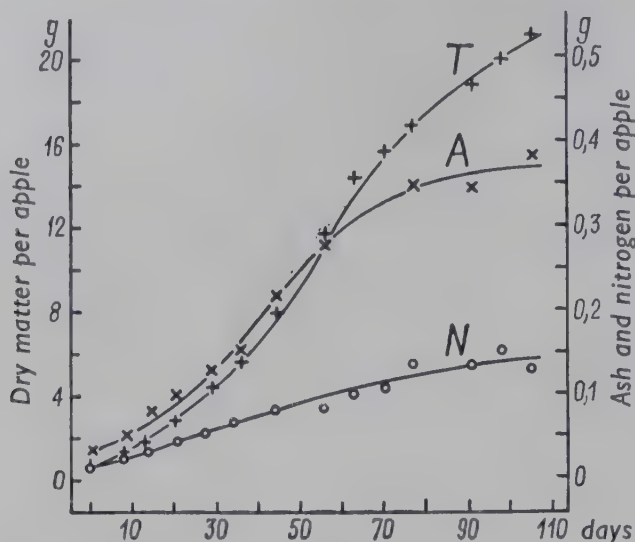


FIG. 234. The total dry matter (*T*), nitrogen (*N*), and ash content (*A*) of apples (var. Dunn's Favourite) during the course of their development. (After Askew, 1935.)

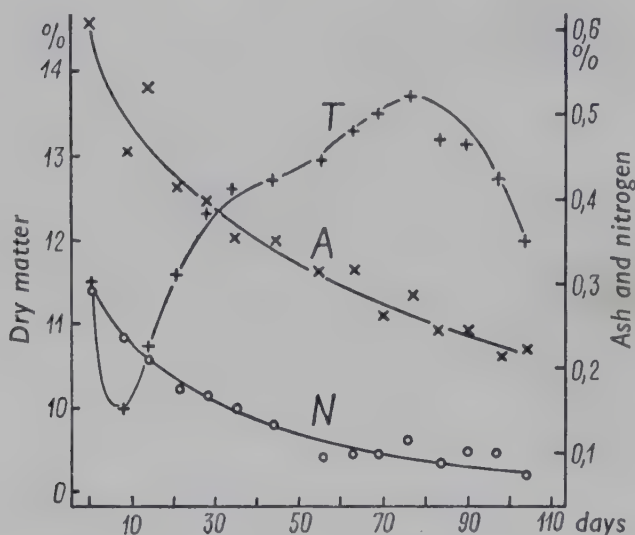


FIG. 235. The percentage dry matter (*T*), nitrogen (*N*), and ash content (*A*) of apples (var. Dunn's Favourite) during the course of their development. The values are given as percentages of the fresh weight. (After Askew, 1935.)

of the substrate. In synthetic nutrient media with very low acid content (less than 0.1% malic acid) the growth rate is greatest with a lower nitrogen (asparagin) content, whereas between 0.15% and 0.3% acidity the relations are reversed (Fig. 238).

The significance of single factors thus varies according to the constellation of the other factors (p. 363) and, therefore, up to the present, it has not been possible to apply the results of laboratory cultures to growing fruits.

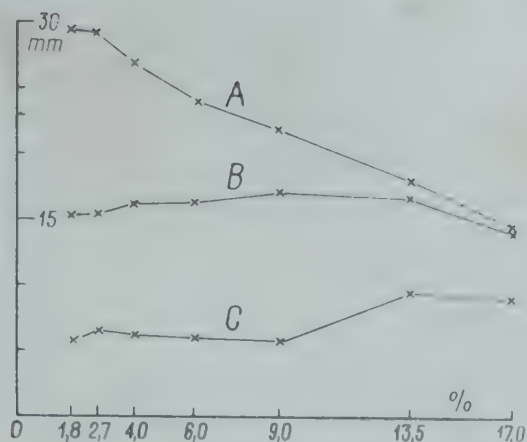


FIG. 236. The radial growth-rate of *Cytosporina ludibunda*, strain CE, in 9 days, with a malic acid concentration of 0.025% (curve A), 0.42% (curve B), and 1.2% (curve C). Abscissae: % glucose. (After Seth, 1934.)

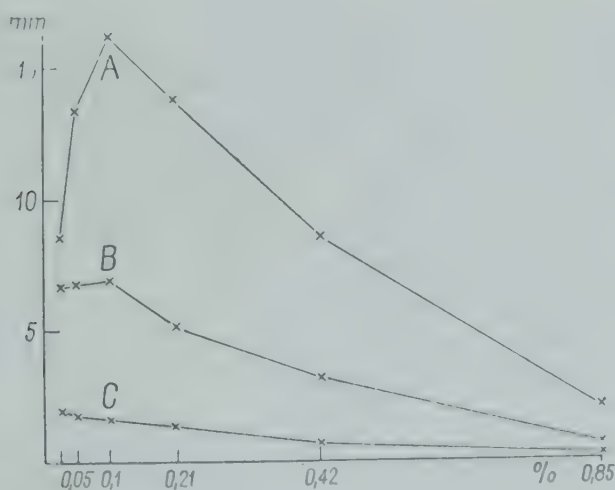


FIG. 237. The radial growth-rate of *Cytosporina ludibunda*, strain CC₂, in 9 days, with a glucose concentration of 2.7% (curve A), 6.0% (curve B), and 17% (curve C). Abscissae: % malic acid. (After Seth, 1934.)

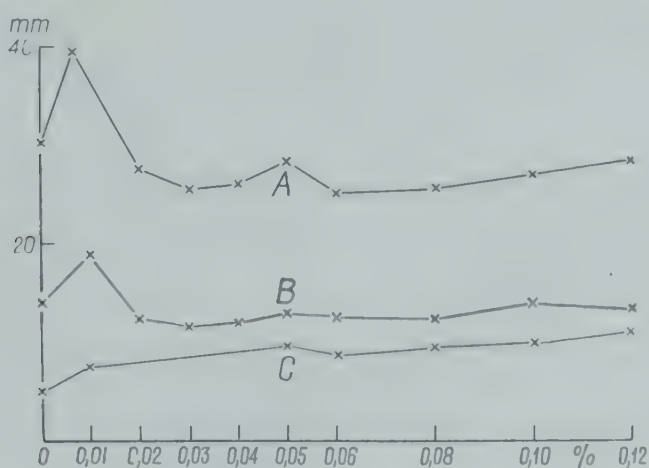


FIG. 238. The radial growth-rate of *Fusarium fructigenum*, strain D, in 9 days, without malic acid (curve A) and with a malic acid concentration of 0.15% (curve B) and of 0.30% (curve C). Abscissae: asparagin content, as % nitrogen. (After Carter, 1934.)

In addition to the changes in resistance to spread during the growth and ripening of the fruit, changes in the level of immunity doubtless also occur, which are regulated by general metabolic conditions. Thus, the respiration intensity of the tomato (Fig. 239) decreases continuously until maturity, then sometimes rises slightly during ripening, but finally falls again when the fruit is ripe enough to pick.

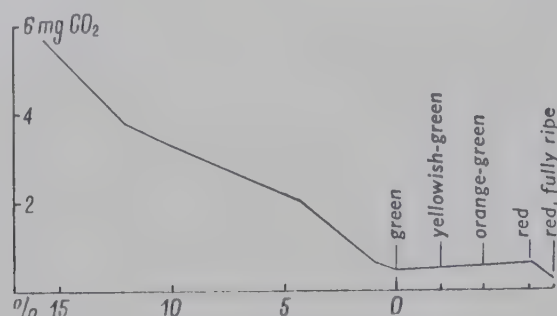


FIG. 239. Respiration rate of tomatoes at different physiological ages. Abscissae: % increase in diameter of fruit per day; when fully grown changes of colour serve as criteria of ageing. Ordinates: amount of CO_2 given and per mg. of fresh weight, per hour. (After Gustafson, 1929.)

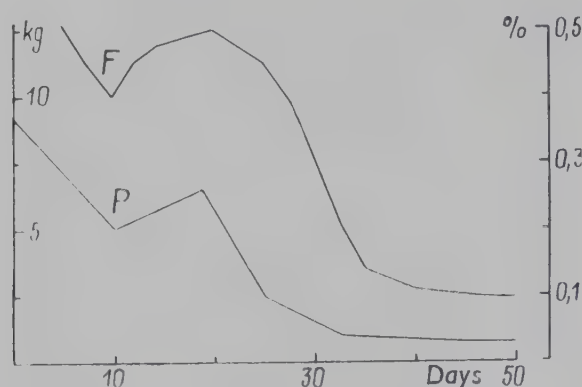


FIG. 240. Softening and pectin degeneration in pears during storage at 4.5°C . Curve *F*: (rigidity) force in kg. which must be applied to a special needle to push it into the fruit. Curve *P*: protopectin content as % of fresh weight. Abscissae: storage time in days. (After Kidd *et al.*, from Paech, 1940.)

These changes also occur in a characteristic manner in stored fruits both after picking and during after-ripening and ageing. Thus, the respiration intensity of pears falls immediately after harvest during a first phase in which starch is being stored, reaches a minimum, and then, due to the greater hydrolysis of reserve materials and of destructible cell-wall components, e.g. pectin, rises to a maximum which, on occasion, may be double the minimum. Subsequently, it falls again until the death of the fruit. This last climacteric increase of the carbon dioxide output clearly indicates the beginning of the real 'ageing' of the fruit. There is no direct relationship to other observable processes, e.g. colouring, pectin breakdown, softening (Fig. 240), since these conform to their own rules.

In the ontogenetic variations of proneness to disease of fruit, as in the

Plasmopara susceptibility of vine-leaves (p. 367), two causal complexes probably overlap one another, viz. an ontogenetic decrease of resistance to spread, consequent upon chemical ripening, and a simultaneous ontogenetic decrease of defence potency, i.e. in the level of immunity of the cells, which will be discussed in the following sub-section.

(d) *Ontogenetic Changes in the Level of Immunity*

Not all plants show ontogenetic variations in their level of immunity. *Dactylis glomerata* remains equally susceptible to *Puccinia graminis* (black rust) throughout its life (Stakman and Piemeisel, 1917). In contrast, for example, to the changes in resistance to penetration, the changes in level of immunity, where they occur, seldom pass from one extreme to the other. Individuals which are completely immune in youth very seldom become highly susceptible in age and vice versa; as a rule, it is only the degree of an already existing susceptibility that becomes modified (although, perhaps, it may sometimes be nullified). Consequently, it is often hard in practice to differentiate between an increase of defence readiness (the 'disinclination' of the organism to serve as host) and a lessening of disease proneness (the 'inclination' of the organism to react pathologically to infection). The phenomena may exist side by side or they may interfere with each other.

All four possibilities of ontogenetic change in level of immunity are represented among the numerous species of host plants and parasites in phytopathology:

- (aa) Youth resistance-age susceptibility,
- (bb) Youth susceptibility-age resistance,
- (cc) Youth and age resistance with susceptibility in middle life,
- (dd) Youth and age susceptibility with resistance in middle life.

(aa) Youth resistance-age susceptibility. This type of ontogenetic change, a lowering of the efficiency of immune reactions with increasing age, is probably the most common in herbaceous plants. As indicated on page 345, viruses cannot attack the growing-point; a similar youth resistance exists in seedlings, which are not susceptible to virus until they begin to form chlorophyll.

In practical crop husbandry this ontogenetic increase in disease proneness becomes apparent in an annual increase in the incidence and severity of disease. The time of the annual epidemic occurrence of some infectious diseases is later than would be expected on parasitological grounds; it does not occur until the cultivated plants have attained their ontogenetic disease proneness. Table LI brings together some results of field experiments on the phase-specific increase of susceptibility in potato plants to late blight. Planting continues from mid-March to mid-August. The first column shows the date of planting, and the following columns give the severity of attack, measured on a scale from 0 (no attack) to 9 (lethal attack). Although the disease was present throughout the field from about 10

August, the lots planted in July were first attacked to an appreciable extent in mid-September. Thus, in the period from mid-August to mid-September, i.e. in the second month of their growth, the plants still retained their condition of developmentally determined internal resistance. In the varieties tested, therefore, *Phytophthora* is an age-disease of the potato plant, but in other varieties no such changes in the level of immunity appear to exist (Beaumont, 1934).

TABLE LI

The relation between the stage of development of potato plants (var. *Eclipse*) and their susceptibility to *Phytophthora infestans*.

(After de Bruijn, 1926)

Date of planting	Severity of attack on									
	August			September				October		
	10	17	25	3	10	17	24	1	8	16
Mid-March	0	0	5							
Mid-April	0	0	5	9						
Mid-May	0	2	6	9	9	9				
Mid-June	0	0	1	4	6	9	9			
Mid-July		0	0	3	4	7	8	9		
Mid-August						3	4	6	8	8

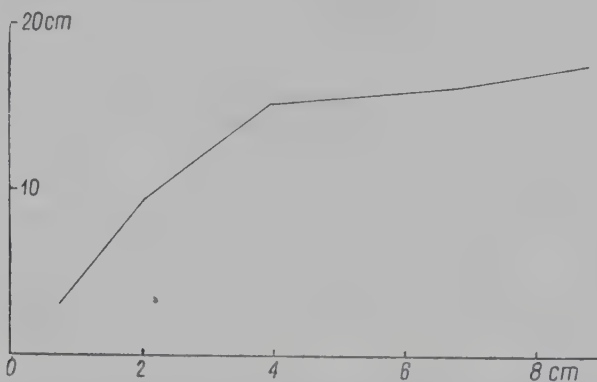


FIG. 241. The phase-specific increase of susceptibility of Weymouth pine to blister rust. Abscissae: diameter of trunk. Ordinates: spread of mycelium after 133 days. The level of immunity decreases asymptotically until the diameter (at chest height) reaches 10–15 cm., which takes about 20–30 years. (After Rhoads, 1920.)

A similar phase-specific increase of disease proneness occurs in the following cases: in plum leaves to plum rust (*Tranzschelia pruni spinosae*), which, for this reason, first appears epidemically in central Europe in September and October; in rose-leaves to black spot (*Diplocarpon rosae*), whose frequency, therefore, also increases towards autumn; in leaf spot of sugar beet (Fig. 122), in which the density of infection shows a similar steep rise in the autumn; and in the attack on the Weymouth pine by blister rust (*Cronartium ribicola*; Fig. 241), in which the mycelium spreads into the cortex of the trunk more quickly the thicker, i.e. the older, the trunk is, &c.

(bb) Youth susceptibility-age resistance. An age-conditioned loss of disease proneness to yellow rust (*Puccinia glumarum*) which an organism possessed in youth has sometimes been observed in cereals. The relations, however, vary greatly according to the variety, the rust biotype, and the temperature during development. Thus, as shown in Table LII, the reaction of Ridit wheat to biotype 31/2 diminishes with age, whereas against biotype Langenstein it does not.

TABLE LII

The influence of the stage of development on the reaction norm of three varieties of wheat to three biotypes of wheat yellow rust (Puccinia glumarum tritici) at an air temperature of 15° C. (After Küderling, 1936)

Variety of wheat	Biotype	Stage of development of host		
		seedling	4-leaf	culm shooting
Ridit	Kitzeberg 31/2	3-4	0-1	0
	Kitzeberg 32	3-4	0	0
	Langenstein	3-4	3-4	3-4
Garnet	Kitzeberg 31/2	2-3	0-i	0-i
	Kitzeberg 32	2-3	i	0-i
	Langenstein	3	0-i	0-i
Hörnings Club wheat .	Kitzeberg 31/2	3-4	3-4	3-4
	Kitzeberg 32	4	4	4
	Langenstein	3-4	0-3	2-3

The difference between youth susceptibility and age resistance can be so great that young cereal plants or young tissues of mature plants may be susceptible to rusts which they are able to resist when mature. Seedlings of particular oat and rye varieties are attacked by wheat black rust (*Puccinia graminis tritici*) forms 9, 21, and 36 whereas older plants are not attacked, and, further, seedlings of barley but not of wheat are attacked by oat black rust (*Puccinia graminis avenae*) forms 6, 7, and 8 (Newton and Brown, 1934).

(cc) Youth and age resistance with susceptibility in middle life. This course of development is also found in some rust susceptibilities. Table LIII gives the data for bean leaves infected with uredospores of bean rust at intervals of 3 days under given external conditions. At first they show an increase in susceptibility evident in the change of reaction type from predominantly 0b to predominantly 3; this is followed by a very sudden reversal between the ninth and twelfth days. At still greater ages of the host tissue, if the rust begins to form teleutospores, the leaves can no longer be infected with the earlier spore forms, the aecidiospores and uredospores: as soon as the tissues reach the stage of 'teleutospore ripeness' they again lose their rust susceptibility.

In cereals a similar decrease during a certain age period can sometimes be observed in the level of immunity to black rust (*Puccinia graminis*) and, to a lesser extent, to crown rust (*P. lolii*; syn. *P. coronifera*). The youth

(seedling) resistance gives place to a certain susceptibility which, in the stage of maturity of the tissues that allows teleutospore formation to occur, changes back again to resistance (Gassner, 1915, 1932). Since the germ tubes penetrate through the stomata, the temporary increase in susceptibility is not, in the first instance, due to a reduction in resistance to penetration or to spread but to a decrease in the physiological defence potential.

TABLE LIII

The influence of age of leaf tissue on reaction type in the bean var. Red Valentine to a given strain of bean rust (Uromyces appendiculatus).

(After Wei, 1937)

Age of leaves after unfolding (in days)	% of leaves with infection type:		
	0b	1b	3
0	54.6	44.7	0.7
3	56.5	43.0	0.5
6	23.8	36.6	39.6
9	21.3	40.6	38.1
12	3.0	13.5	83.5
15	0.6	0.5	98.9
18	0.2	0.2	99.6
21	0.4	0.5	99.1

The relationships, however, as always with the rust fungi, are in reality very complicated and our conclusions possess only a general validity. Wheat as a botanical species (or species group) does not, during its growing period, necessarily become more susceptible to black rust; any statement is true only for given wheat varieties in association with given biotypes of black rust. For instance, we have already seen an example in (*bb*) in which this relationship was reversed. In addition, the assessment of the susceptibility sometimes differs according to whether the criterion used is the incidence of attack (the infection frequency, the number and size of infection sites or rust pustules) or the infection type (the reaction type of the host plant, p. 246).

(*dd*) Youth and age susceptibility with resistance in middle life. An ontogenetically determined susceptibility in youth and in age, associated with a heightened resistance 'in the prime of life', is occasionally found in wheat varieties in relation to certain biotypes of brown rust (*Puccinia triticina*). In them, when the plant begins to shoot and flower, the high youth susceptibility (attack type 4, Table LIV) is sometimes replaced by marked resistance (attack type 0); when older leaves reach 'teleutospore ripeness' they again show an increased susceptibility.

The fact that resistance to the different rusts and rust biotypes appreciably changes during the development of one and the same cereal plant—diminishing with one rust or rust biotype according to type (*aa*), increasing with another rust according to type (*bb*), and, with a third, varying according to type (*cc*)—shows that changes in reaction norm, i.e. in

level of immunity, and not changes in the resistance factors, are mainly concerned here. Host plants showing such ontogenetic changes in their level of immunity usually exhibit a similar range of sensitivity to external influences (nutrition, temperature, &c.), shortly to be discussed.

TABLE LIV

The ontogenetic differences in the susceptibility of some varieties of wheat to biotype 15 of wheat brown rust (Puccinia triticina). (After Vohl, 1938)

<i>Developmental stage</i>	<i>Marquis</i>	<i>Marquillo</i>	<i>Thatcher</i>	<i>Hope</i>
Yellow ripe. .	1	2	2	1
Milk ripe . .	1	1	1	0
Ear shooting .	0	0	0	0
Culm shooting .	1	1	1	2
Tillering . .	3	3	3	4
Seedling . .	4	4	4	4

These ontogenetic changes in level of immunity raise difficult problems for the plant breeder. In view of the climatic conditions and the rust races within a given area of cultivation, what kind of resistance against rust should he aim to achieve; should it be resistance during tillering (which, in winter wheat, takes place partly in the autumn of the previous year), or during the shooting of the ears, or during the period of milk-ripeness?

Moreover, these changes make the laboratory testing of new varieties for rust resistance extraordinarily difficult. It is not enough merely to inoculate seedling plants in greenhouses with a number of rust biotypes; costly field-scale experiments are necessary but, even in these, the ontogenetic change in level of immunity will be obscured by the changes in disposition caused by temperature variations, &c., during the year, so that the results vary from one area of cultivation to another.

2. *The Influence of the Vitality of the Host on its Disease Proneness*

By vitality, life energy, or *élan vital*, we understand the intensity with which an organism as a whole makes demands on and reacts to the outer world. It constitutes one of the bases of disease proneness.

All the factors which alter the disease proneness of the host plant actually accomplish this through the vitality of the host organism. So far as these factors have been isolated and can be considered independently (external temperature, illumination, particular nutrients, &c.) they will be discussed singly in the following sub-section. In regard to them we know the initial link of the reaction chain (the given variable environmental factor), the penultimate link (the changing vitality), and the terminal link (the similarly changing disease proneness); but, at present, the intermediate links remain inaccessible to us.

In other changes of vitality it has not yet been possible to resolve the initial link, the environmental complex, into its components; only the penultimate link, the altering vitality, and the terminal link, the altered

disease proneness, are understood. Some of these examples, in which the decisive environmental factors cannot yet be defined, will be discussed in the present sub-section.

The direction in which the disease proneness of the host is altered varies according to the eusymbiotic or parabiatic character of the parasite-host-relationship, and is usually paradoxical. In eusymbiosis, a lowering of the vitality of the host leads to a heightened susceptibility, whilst a strengthening of the host leads to a diminished susceptibility; in parabiosis, on the other hand, a weakening of the host results in diminished susceptibility and a strengthening of the host leads to a heightened susceptibility. In eusymbiosis, therefore, low vitality means a severe attack, whereas in parabiosis it means freedom from attack (p. 297).

Many examples of the first case, heightening of disease proneness due to lowering of the vitality of the host, are familiar in both medicine and plant pathology. Those parasites which, usually, are able to colonize a host only when its vitality is abnormally reduced are known as weak parasites (e.g. *Botrytis cinerea*, *Macrosporium* spp., *Cladosporium* spp., black moulds of agricultural plants, &c.); they belong by nature to the facultative parasites.

The more intensive the cultivation, the less stable is the vital equilibrium of the plants concerned, and the greater the risks due to an induced disposition. Thus, in the plant kingdom, there are diseases of domestication analogous to tuberculosis in man and cattle. The gardener, therefore, has to reckon with susceptibility due to weakness more than has the farmer. The greenhouse creates so many of these special disposition conditions that true greenhouse diseases arise (Bewley, 1928).

Evidently another group of these obscure environmental complexes forms the climate of cities; this renders deciduous trees lining the streets prematurely susceptible to *Nectria* attack, and the conifers to leaf cast fungi. Exhaust gases of combustion engines, street lighting, chimney smoke, and the extremely dry habitat (intensive radiation, combined with lack of water and air for the roots due to their being covered by a hard pavement) create an artificial local climate to which the inner organization of the trees can no longer adapt itself. Asphalt plants do not usually attain any great age.

In orchard and forest practice, as in gardening, the life energy of individual trees is carefully fostered, because in this case cultural treatment and observation can be carried on for decades. Fig. 242 shows how greatly the disease disposition of plum trees to silver leaf fluctuates with the vitality during the growing period. In the inactive period, February–April, 100% of the twig infections lead to characteristic outbreaks of disease; with the reawakened vitality in May the power of defence increases (p. 342), reaches a climax in June–July, and diminishes again towards autumn. An alteration of the general living conditions of trees by cultural operations works in the same direction but is more difficult to follow.

A similar situation is present in relation to the supply problem in forestry. The genetical breeding of trees of high quality produces results only after

very many years, so that, in the meantime, afforestation must be carried out with seed of local races of high quality from districts with similar climatic and edaphic conditions. Yet many forest trees show a remarkably low capacity to adapt their nutrition and developmental rhythm to the new conditions of life; their vitality is lowered in the new surroundings and, in consequence, they are more inclined to suffer from infectious diseases than they were in their native habitat.

In the experimental gardens at Eberswald 1-year-old pines from different seed areas were planted out, and after 2 years the following percentages

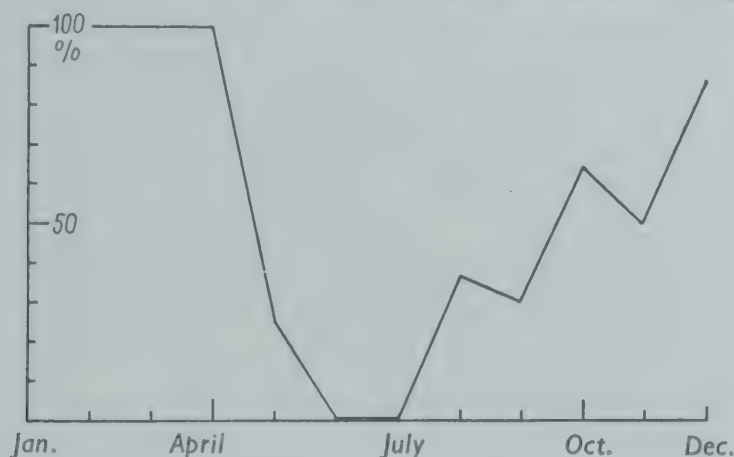


FIG. 242. The susceptibility of plum trees to *Stereum purpureum* when inoculations were made throughout the course of a year. Abscissae: time of infection. Ordinates: number of diseased trees. (After Brooks and Moore, 1926.)

had been killed by needle leaf cast (*Lophodermium pinastri*): 87% of stock from the Rhine Palatinate, 86% from Auvergne, 85% from Belgium, 78% from the Mark, 57% from Johannisberg heath, 52% from Scotland, 35% from Courland, and 25% from the Urals (Haaek, 1911). In similar experiments in northern Switzerland (Eglisau; Burger, 1931) it was just the pines from the neighbouring Swiss Alps that suffered severely from needle cast, whereas the pines from south-western Germany and the south of France suffered only moderately or slightly, and the pines from East Prussia, Sweden, Norway, and the Urals only very little or not at all. Similar results were obtained in Sweden with the snow cast of firs, *Phacidium infestans* (Schotte, 1923).

The forester therefore concludes that East Prussian seed is 'better suited to the locality' than Alpine seed and, in consequence, is more resistant to leaf cast; the botanist also explains the causes of these great variations in susceptibility by the equally general conception of a depression of vitality.

In contrast to the infectious diseases so far discussed, in the parabiotic parasite-host association a lowering of vitality leads to a diminution of infection- and disease-susceptibility, and a raising of vitality leads to an increase of infection- and disease-susceptibility.

In certain cases, as in the influenza epidemic of 1918, the physician also assumes a similar relationship. This naturally cannot be proved experimentally, but only established statistically on the basis of the differential

mortality of physically strong and physically more frail occupational classes. But the criticism remains that the physically strong men only receive medical treatment too late and then succumb to complications.

In botany a correlation between the vitality of the host and its proneness to disease exists in certain infectious diseases of tubers, fruits, &c. In potatoes the optimum temperature for yield (Fig. 255) is practically the same as that for scab attack (Fig. 259). It is the host, after all, which responds to the pathogenic stimulus with morbid growths. In this instance the greater the vitality the more effective is the pathological reaction.

TABLE LV

The influence of vernalization on bunt infection of wheat.
(After Lasser, 1937)

<i>Variety of wheat</i>	<i>Vernalized %</i>	<i>Not vernalized %</i>
Ridit	3.4	5.7
Heils Dickkopf . .	11.7	38.2
Carsten V	7.1	54.9
Panzer III	20.2	66.2

This is also true in certain infections of foliage leaves, especially rust diseases. One of the few exceptions is the spruce needle rust (*Chrysomyxa abietis*) which predominantly attacks over-topped spruce trees.

Table LVI and Fig. 243 give the means of thirty-two experiments showing the relation between planting distance and rust attack in oats (black and yellow rusts). By increasing the spacing, each individual plant obtains more soil, air, and light, it tillers more freely (number of panicles), and yields more (straw and grain yield). But at the same time its susceptibility to rust increases, the rust-infected proportion of the leaf-surface rising from 11.8 to 33.2%.

Clearly it is worth while for the farmer to allow his oats to grow luxuriantly by giving suitably balanced fertilizers, since the curve of yield in Fig. 243 rises more steeply than the curve of rust infection. The rust attack increases about threefold and the grain yield per plant about one-hundredfold. In this instance, therefore, the heavier rust attack is of no economic importance, but from the standpoint of biological theory, the knowledge that in certain diseases an increased vitality conditions a heightened susceptibility is important. This circumstance must be taken into account in controlled experiments on rust infection; if the growth of the experimental plant (pot plant!) is not optimal, the results are often erroneously negative.

The experiments on the influence of vernalization on the disease disposition of cereals may, perhaps, be explained in the same way (Lasser, 1937). Vernalization is a method of altering winter cereals in the seedling

stage: the seed is moistened in a given way, germinated at 12–15° C. and, as soon as the seedling has broken through the testa (3 weeks for winter barley and 5 weeks for winter wheat), is kept at about 3° C. This procedure decisively alters its germination so that winter cereals shoot when sown in spring. But, at the same time (Table LV), smut susceptibility is also

TABLE LVI

Relationship between planting distance and rust attack in oats
(After Raines, 1922)

Distance apart (cm.)	No. of panicles per plant	Relative yield per plant		Leaf surface attacked by rust (%)
		Straw	Grain	
2.5	1	1	1	11.8
5.0	1.1	3.6	4.6	15.0
7.5	1.3	7.8	12.3	17.8
10	2.0	11.8	20.3	20.9
15	4.2	28.2	44.0	25.4
20	6.5	43.9	66.5	27.7
30	11.2	84.8	103.2	33.2

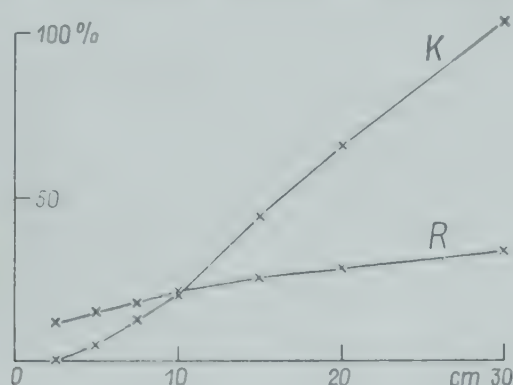


FIG. 243. The increase in grain yield (curve K) and rust attack (curve R) with increasing distance between oat plants. (After Raines, 1922.)

greatly reduced. It is uncertain whether, in this case, there is a true increase of resistance or only an apparent increase due to the seedling out-distancing infection because of its accelerated development.

3. The Influence of Nutrition on the Disease Proneness of the Host

By nutrition we refer exclusively to mineral salt nutrition; the remaining cultural factors, water supply, photosynthesis, &c., will be examined separately.

The present sub-section is classified under six headings:

- the specificity of the change in disposition,
- the degree of changeability in disposition,
- the instability of the disposition,
- the trend of the change in disposition,
- the changeable elements of the disposition,
- the effects of particular nutrients.

(a) The specificity of nutritional change in disposition. The changes in the disease susceptibility of plants due to nutrition are generally non-specific. It is not merely the susceptibility to a given infectious disease that is altered but likewise the susceptibility for a number of other diseases. The latter may, therefore, be grouped according to the nature of the factor concerned.

(b) The degree of nutritional changeability in disposition. In man the nutritionally conditioned variations in disease disposition are very great because, for psychological and sociological reasons, human food is extremely diverse. One has only to think of the great danger of infection resulting from severe under-nourishment (famine) or from partial under-nourishment (e.g. C-avitaminosis, scurvy) or from excess nourishment (e.g. habitual greed). Clearly, similar errors in nutrition can also arise in plants, but they are not of current interest to the plant pathologist. For him it is not a question of turning unsuitable hosts into suitable ones but, within the range of a more or less normal (non-pathological) nutrient supply, to trace the change in susceptibility due to an unbalanced, one-sided food supply. Corresponding to this limitation in the causal range of variation the changes induced in the experimental plants are much slighter than those occurring in man.

(c) The instability of the nutritionally determined disposition. Both the degree and the readiness with which the disease disposition can be altered by nutrition varies according to the pathogen and the host. The susceptibility of potatoes to wart disease is little influenced by manuring, whereas their susceptibility to *Phytophthora* is greatly affected. Smut attack on cereals is only slightly influenced by nutrition, whereas attack by rusts, mildews (Erysiphaceae), and stripe diseases (*Helminthosporium* spp.) is affected.

(d) The trend of nutritional change in disposition. This varies according to the pathogen-host association and is often paradoxical. A weakening of the host plant favours attack by eusymbiotic parasites, a strengthening of the host favours attack by parabiogenic parasites (p. 297): impoverished cereals or those with unbalanced manuring are attacked more severely by *Helminthosporium*, whereas well-nourished plants are attacked more severely by rust fungi. Deficient plants are generally more resistant to rust fungi than are well-nourished or over-fed plants. Hence, with intensification of agriculture (e.g. heavy manuring to increase yield) the danger to the plants from rust attack increases.

(e) The changeable elements of nutritionally conditioned disposition. Unbalanced nutrition affects the occurrence of disease in plants and man in three ways: it influences the infection proneness, the clinical picture, and the course of the disease. Accordingly, the place at which it affects the resistance system of the host plant differs; its influence may be either direct (immediate) or indirect (mediate).

The direct influence of unbalanced nutrition makes itself felt either mainly structurally (through factors of resistance) or mainly functionally

(in the adequacy of defence reactions). However, the two types of effect are sometimes coincident, as in the case of faulty nitrogen or potash supply (Table LVII).

TABLE LVII

*The commonest changes in disease proneness due to unbalanced nutrition.
(Mainly after Roemer et al., 1938)*

Function	Nitrogen		Potassium		Phosphoric acid	
	Deficiency	Excess	Deficiency	Excess	Deficiency	Excess
Development of the resistance factor . .	Increased	Decreased	Decreased	Increased	Variable	
Capacity for defence reaction .	Increased	Decreased	Decreased	Increased	Variable	
Water content .	Low	Very high	Low	Very high	High	Low
Sugar content .	High	Low	High	Low	Very low	Very high
Starch content .	Irregular		Low	Very high	High	Low
Osmotic pressure of the cell sap	Low	High	Very low	Very high	High	Low
Formation of resistant tissues	Moderate	Slight	Slight	Very great	Slight	Very great

An alteration of the structural resistance (viz. the formation of scaffolding substances) takes place, for example, in potato tubers against wet rot bacteria. In the first place the latter destroy the middle lamellae, and then the cell formation is disrupted and the individual cells degenerate into a rotten, pulpy mass. Table LVIII shows that where nitrogen and potash supply are faulty a certain correlation exists between the dissolution of the middle lamellae by *Bacillus atrosepticus* and the number of rotten tubers occurring spontaneously in a clamp. But a disturbance in the phosphorus supply does not have the same effect; thus (in spite of the greater solubility of the middle lamellae) this phosphorus imbalance must condition a higher functional stability.

An alteration of the functional resistance of the host was found, for instance, by Böning (1930, 1933) for the susceptibility of tobacco to wild fire (p. 18): excess nitrogen or potash deficiency increased the attack, whereas low nitrogen or high potash decreased it; phosphorus deficiency acts in a similar way to nitrogen excess, whereas excess phosphoric acid hardly affects the form of attack. However, no clear correlation seems to exist between the nitrogen content of the leaves and their disease resistance, so that their capacity for functional defence is evidently decisive.

The indirect effect of unbalanced nutrition on the disease disposition of the host shows itself, in one way amongst others, in relation to growth-rate: cereals over-manured with nitrogen bloom later, those over-manured with phosphorus bloom earlier. The conditions of infection and the ontogenetic susceptibilities are correspondingly altered; black rust, for example, generally 'prefers' the more mature stages of development of the host.

(f) The effects of particular nutrients. Nitrogen occupies a central

position among the several nutrients which appreciably influence the disease disposition of the host. Excess of nitrogen relative to phosphorus and especially to potassium (Richards and Templeman, 1936) accelerates development, 'weakens' the tissues, delays their maturation, and thus prolongs their life. Excess nitrogen nutrition also causes 'rank' growth of cultures (fruit growers and foresters refer to disease-susceptible wood as having 'grown pampered') and generally reduces disease resistance (Table LVII). Nitrogen deficiency, on the other hand, accelerates the physiological ageing of leaves: thus, leaves produced at the same time but at different levels of nitrogen nutrition differ in their physiological ages.

TABLE LVIII

The influence of nutrition on wet rot attack of stored potatoes, industrial variety. (After Fehmi, 1933)

<i>Manuring</i>	<i>Solution of middle lamellae (in days)</i>	<i>Spontaneously rotted potatoes</i>
		%
Normal manuring	9	5.9
Nitrogen		
Excess as calcium nitrate . .	8	17.5
Excess as ammonium sulphate .	8	20.8
Deficiency	9	2.8
Potash		
Excess as Kainit	9	8.4
Excess as 40% calcium salts .	8	11.4
Deficiency	8	12.1
Phosphoric acid		
Excess as superphosphate . .	8	7.1
Excess as 'Thomas meal' . .	7	6.8
Deficiency	7	6.9

The opposed effects of excess nitrogen manuring on the rate of development and on disease resistance are shown in Fig. 244. The plants were grown in sand culture; both received a standard dose of phosphorus and potassium, but that on the right was also given 2 g. of ammonium sulphate. The stems of both were inoculated with spores of the wilt-causing fungus, *Verticillium albo-atrum*. Fig. 244 *A* shows the extent to which the development of the plant with excess nitrogen on the right has surpassed the deficient plant on the left, but the former also shows marked symptoms. Later (Fig. 244 *B*), this plant succumbed completely to the disease, whereas infection in the deficient plant failed.

However, this disease furthering effect of excess nitrogen is not universally valid; in the root nodules of Leguminosae (p. 281) nitrogen deficiency creates the disposition to disease.

Two examples, *Phytophthora* susceptibility of potatoes and rust susceptibility of cereals, may be used to illustrate more explicitly the significance and the effect of the nitrogen factor.

Phytophthora infestans does not use as its main source of energy the carbohydrates of the host tissues, but the nitrogen-containing fractions, especially the amino-acids (Lepik, 1939; Hagenguth and Griesinger, 1943). Only a small part of the starch which the tuber loses in the course of the disease is used by the fungus, most of it is respired away by the host or turned into sugar by secondary processes (Table LIX). It is probable, therefore, that the nitrogen metabolism of potato plants not only influences



FIG. 244. The effect of N nutrition on the rate of development of tomato plants and on their resistance to *Verticillium albo-atrum* (wilt disease). *A*, plants without nitrogen (—) and heavily manured with nitrogen (NN), 4 weeks after infection. *B*, the same plants 8 weeks after infection. Explanation in text. (After Donandt, 1932.)

their readiness to react (p. 309) but also, to a very great extent, their suitability to act as a substrate for the parasite.

TABLE LIX

The mean carbohydrate and nitrogen content (in % dry weight) of healthy and Phytophthora-infected potato tubers. (Combined from two experiments by Lepik, 1929, 1939)

Substance	Healthy portions of tuber	Diseased portions of tuber
	%	%
Starch . . .	70.3	64.8
Saccharose . .	1.7	3.0
Invert sugar . .	0.04	0.11
Total nitrogen .	1.17	0.91
Crude protein . .	7.0	5.5

Alten and Orth (1941) grew potatoes of the variety Voran in synthetic nutrient solutions containing differing amounts of nitrogen and potassium. The content of the total nitrogen in the plants varied with the ratio of K:N supplied (Table LX). In solutions low in nitrogen (100 mg. per litre), the total nitrogen content of the plants decreased with increasing

potassium supply, and the values of protein-, non-protein-, and α -amino-acid-nitrogen decreased similarly. On the other hand, if the nitrogen supply was increased, the production of nitrogenous substances did not continue to rise with increasing potassium supply but remained remarkably constant. Hence, increasing nitrogen supply accompanied by simultaneous increase of potassium supply led to an upper limiting or maximal value in the potato plants. A clear answer cannot, therefore, be given to the question whether or not nitrogen or potassium application is favourable to the production of nitrogenous compounds in the potato plant, thereby increasing or decreasing its susceptibility to *Phytophthora*, since the effect of nitrogen treatment depends on the amount of potash available and vice versa.

TABLE LX

Influence of potassium and nitrogen nutrition on the nitrogen metabolism of the potato plant. N and arginine in % dry weight. (After Alten and Orth, 1941)

mg. K ₂ O/l. solution	100 mg. N/l. of nutrient solution					400 mg. N/l. of nutrient solution				
	% Total N	% Protein-N	% Non-protein-N	% α -amino-acid-N	% Arginine	% Total N	% Protein-N	% Non-protein-N	% α -amino-acid-N	% Arginine
25	3.9	2.5	1.4	1.0	1.4	3.9	2.5	1.4	0.8	1.9
50	3.4	2.1	1.3	0.9	1.5	3.9	2.5	1.4	0.8	1.9
100	3.1	2.0	1.1	0.7	1.6	4.0	2.5	1.5	0.7	2.0
200	2.7	1.8	0.9	0.6	1.8	4.2	2.6	1.6	0.7	2.1
400	2.3	1.5	0.8	0.5	1.9	4.4	2.6	1.8	0.7	2.2
800	2.4	1.6	0.8	0.4	1.9	4.2	2.6	1.6	0.7	2.3

Moreover, the relation between rising nitrogen content and rising *Phytophthora* susceptibility is not solely quantitative, but is affected by the nature of the several nitrogen compounds present. Most amino-acids are excellent nutrient materials for *P. infestans*, but arginine (δ -guanidino- α -amino-valeric acid), one of the most frequently occurring amino-acids, is very poisonous to it (though not, for instance, to *Fusaria*) even in as low a concentration as 1:1,000. But it is just the arginine content of the potato plants which increases in the foliage and, to a lesser extent, in the tubers, with increased supply of nitrogen and potassium (Table LX). Hence, within the total nitrogen metabolism of the potato plant, a quantitative change occurs in the nitrogenous components which is adverse to the growth of *Phytophthora* and tends to counteract the quantitative beneficial effect. Hence, that treatment is best adapted to reduce the *Phytophthora* susceptibility of potatoes which diminishes the total-, protein-, non-protein-, and amino-acid-nitrogen content of the plant whilst increasing its content of arginine.

It is clear that arginine alone, through its inhibitory effect, does not determine quantitatively the susceptibility of potatoes to *Phytophthora*. But the example shows how a single nutritive change (e.g. in the nitrogen metabolism of the host plant) can produce antagonistic effects on the fitness of the plant to serve as a host to parasites, so that a simple correlation between total nitrogen level and *Phytophthora* susceptibility is automatically ruled out.

As in the case of nitrogen metabolism and *Phytophthora* susceptibility of potatoes so in the second example, the cereal rusts, the nitrogen does not produce its effects independently, as such, but operates in relation to other nutrients and to assimilation.

If the plants have sufficient potassium, the application of nitrogen favours metabolic syntheses: chlorophyll content, assimilation, transpiration, and protein content of the leaves all increase. There is generally a parallel rise in rust susceptibility; the greater the excess of nitrogen over potash and phosphoric acid, the steeper is this rise. In addition, the nitrogen effect depends on the relative balance of potassium and phosphorus (Gassner and Goeze, 1934; Gassner and Franke, 1934).

The heightened rust susceptibility is due to a decrease in the reaction proneness and the reaction capacity of the host plants. Spontaneous necrogenic variegation appears round the sites of infection (Table LXI). A necrogenous abortion of the tissues may take place later, but usually not to the same extent as occurs spontaneously in nitrogen deficient plants. In accordance with this weakening of the protective necrogenous reactions, the reaction type moves up in the scheme of disease classification (Table LXI).

These alterations of reaction type are, however, valid only for cereal varieties of intermediate susceptibility. Highly susceptible varieties remain highly susceptible at all levels of nitrogen, and immune varieties remain immune. Again, these alterations are possible only in varieties with a labile infection type, those with a stable infection type remain indifferent to changes in nitrogen nutrition. This explains a number of the contradictions in the literature.

Some of the other features by means of which the rust disposition of the host plant can be estimated are quite unaffected by variations in the nitrogen supply; others are altered in the same way as the reaction type, and still others in the opposite direction. For example, as a rule, the incidence of attack (severity of attack) rises, like the reaction type, with increasing nitrogen nutrition but, under some conditions, it changes in the opposite direction and there is a fall instead of a rise. In this last case, therefore, differences in nitrogen supply have directly opposite effects on host resistance at different stages of infection.

The incubation period and the pre-sporulation period can also be altered by improved nitrogen nutrition, but to different extents. In Table LXI the incubation period is either not prolonged at all or only for one day,

whereas the time to fructification is regularly increased by at least two days. But the significance of this extension is debatable. Judging by the example of *Erysiphe* given on page 247, one might assume that the resistance of the host plant had increased since the parasite needed longer to produce spores. But it is possible to draw an opposite conclusion: the host does not react hyper-sensitively and, therefore, does not compel the parasite to a premature formation of uredosori, i.e. the host has become more susceptible. This example provides a further demonstration of the difficulties encountered in the interpretation of any given factual situation. If an author restricts his attention exclusively to one factor, e.g. frequency of attack, type of reaction, incubation period, or time to fructification, his conclusions as to the effect of increasing nitrogen supply may be contradictory.

TABLE LXI

Influence of nitrogen nutrition on susceptibility to brown rust in two moderately resistant wheat varieties. (After Gassner and Hassebrauk, 1931)

<i>Wheat variety and behaviour of the rust</i>	<i>No nitrogen</i>	<i>Normal nitrogen supply</i>	<i>Fivefold supply of nitrogen</i>
Rümkers early Sommerdickkopf:			
Incubation period in days	4	—	4
Time to sporulation in days	7-8	—	9-10
Symptoms 7 days after infection	Severe chlorosis with necrotic zones. Scattered pustule formation	As with nitrogen deficiency	Light green, weak chlorosis. Pustules visible through surface
Reaction type 12 days after infection .	3-2	3	4
Berkners Continental:			
Incubation period in days	3	—	4
Time to sporulation in days	7	—	9
Symptoms 7 days after infection	Pronounced yellow mottling and beginning of necroses. Scattered eruption of pustules	As with nitrogen deficiency	Yellowish green, soft mottling. No necroses. Pustules only slightly visible through surface
Reaction type 12 days after infection .	3	3-4	3-4

The difficulty is emphasized still further by the fact that although in the cases so far discussed increasing dosages of nitrogen favour rust infection, this outcome is not invariably found (or, at least, not always in the same manner) in all strains of the same rust species. For instance, in the wheat varieties Carina and Brevit, susceptibility to the brown rust biotypes 14 and 20 is increased, this being in agreement with the general

rule as shown in Table LXI, whereas it is decreased to biotypes 13, 19, and 31 (Hassebrauk, 1939).

A similar decrease in susceptibility to particular rust biotypes with rising doses of nitrogen occurs in certain varieties of beans: in the variety Red Valentine (Table LIII), belonging to the intermediate reaction type Xb towards a given rust biotype, the incidence of attack is heightened by increasing nitrogen supply, whereas the reaction type falls to a lower class. With lack of nitrogen and excess potassium the relative proportion of type 3 infections is increased but, under the reverse conditions, this type is largely replaced by type 1b (Wei, 1937).

Nothing is known about the chemical basis of the relations between nitrogen metabolism and rust susceptibility within the host plant. It has often been suggested that proteinaceous compounds have a simple detoxicating action or an otherwise protective effect, but this view leads to a gross oversimplification of the problem. The nitrogen compounds in the protoplasm surely play a double role, a passive one in the immediate nutrition of the parasite (if they are suited to it), and an active one as a link in the chain of anti-infectional and antitoxic defence reactions. The interplay of forces between parasite and host which governs their relationship is so delicate and so infinitely complicated, especially in the rust fungi, that a simple correlation between protein content and rust susceptibility is not to be thought of. Quantitative or crudely qualitative chemical analyses of the nitrogenous compounds explain neither the differences in susceptibility of host varieties to the same strain of rust, i.e. varietal susceptibility, nor those of the same host variety to different rust strains, i.e. the specific susceptibilities of a given variety.

Hence, no simple explanation can be adduced for the effect of varied nitrogen supply on rust susceptibility in cereals. In all probability it will still take generations of thorough cytopathological investigation before we shall be able to view these problems clearly.

Compared with nitrogen the remaining mineral nutrients are of only minor importance in relation to the problem of susceptibility.

The action of potassium is generally opposite to that of nitrogen, reduced supply having the same effect as a relative excess of nitrogen, and excess potassium having a comparable effect to relative lack of nitrogen. Cereals grown under conditions of potassium deficiency and only moderate nitrogen supply show maxima in their chlorophyll and protein contents, and in their assimilation and transpiration rates (Gassner and Goeze, 1933). With increasing potassium supply all these values tend to fall. Yet the nitrogen factor is the more important one since the potassium effect can be eliminated by excess nitrogen: then maximal assimilation rate and protein content of both cereals and potatoes (Table LX) are found to coincide with high and no longer with low potassium supply.

A similar contrast is found in the effect of potassium on disease susceptibility as in that on metabolism: increasing potassium supply generally raises the level of disease resistance, this being the reverse of the nitrogen

effect (Table LVII). If nitrogen and phosphorus supply are varied simultaneously with increases in potassium, the latter becomes more disease inhibiting, up to a certain limiting value, the more it is in excess in relation to nitrogen and phosphorus. Apart from this, its effect depends also on the balance of nitrogen to phosphorus present.

In contrast to the effects of nitrogen and potassium, the action of phosphorus on disease resistance is not altogether unequivocal; it depends to a very large extent on the coincident potassium and nitrogen levels. With a relative deficiency of phosphorus the host plants are usually weakened, and become more susceptible to eusymbiotic parasites. The opposite effect may, however, result with parabiogenic strains of rusts: the tissues may become hyper-sensitive to the parasite, show a quick necrotic reaction, and thus remain free from attack.

When phosphorus is present in excess relative to potassium and nitrogen, it generally heightens disease resistance. But again, in the case of the rust fungi the opposite effect may ensue, and susceptibility may be increased by plentiful phosphorus supply accompanying heavy application of potassium and nitrogen. This effect is large enough to eliminate completely any rise in rust resistance due to excess potassium. Hence, it is impossible to combine the rust-inhibiting effect of potassium with that of phosphorus, since these two substances antagonize one another if used simultaneously.

Of the other mineral nutrients, lime may have a twofold effect: a direct effect as Ca-ion and an indirect one through the soil reaction. Excess calcium enhances resistance to spread by contributing to the hardening of tissues. Silica also generally increases disease resistance, e.g. to powdery mildews in barley, oats, and cucumbers (Wagner, 1940). For the effects of anions Becker-Dillingen's *Handbuch der Planzenernährung* (1934, 1943) should be consulted; for trace elements the work of Scharrer (1941).

It will, of course, be clear that these brief data concerning the influence of mineral nutrition on the disposition of all our cultivated and economic plants to all possible infectious diseases can possess only conditional validity. The response of one host only, e.g. a wheat variety, to all the races of its parasites is so complex, and the number of possible combinations of the variable nutritional factors is so great, that for every example a counter-example can be found. In addition, the mode of reaction of the host alters in the course of individual development. For instance, at first the host organism may react to excess nitrogen as if to phosphorus deficiency, later as if to potassium shortage (Böning, 1930; for *Pseudomonas tabaci* and *Cercospora nicotianae*).

Thus, every variety of cultivated plant possesses, to some extent, its own nutritionally determined disposition to each separate infectious disease. In many cases the grower has already learned empirically how to take this into account. For the biologist it constitutes an almost unlimited field for research.

4. *The Influence of Environmental Temperature on the Disease Proneness of the Host*

The human organism possesses autonomous temperature regulation and, therefore, for all practical purposes, lives at a constant internal temperature. Civilized man has also withdrawn himself to a great extent from the influence of external temperature; he clothes himself, heats or cools his living-rooms, and thus creates for himself a suitable microclimate. Thus, temperature conditions of the environment may well affect his general tone, raise his vitality by harmonious alternations, or debilitate him in the tropics, but his chemical constitution and his functions are altered only to a very slight extent. Consequently, his susceptibility remains fundamentally the same over a wide range of external temperatures. Only by way of changes in vitality (e.g. general debility in the climate of tropical lowlands) or by definite injuries (freezing, &c.) does the outer temperature directly affect the occurrence of disease in man.

Conditions are very different in plants. The plant body has no characteristic temperature of its own but passively adopts that of the environment. The rhythm of its growth is cyclic and is controlled by the rhythm of the seasons. Sun and shade, day and night, locality, local climate, and altitude determine the varying temperature levels at which its chemical changes will occur. Hence, its chemical constitution, its reactivity, and its disease proneness are far more influenced by external temperatures than is the case in man.

Because of this lack in the host of a temperature of its own, in infectious diseases of plants in contrast to those of man, the micro-organism, at least after infection, becomes subject to the external temperature conditions under which the macro-organism lives. Hence, we are obliged to divide the totality of the manifold thermally controlled relations, actions, and reactions existing between the two partners into fractional problems in a more or less arbitrary manner, although these moieties overlap in many ways. Hence, the analysis of the thermally determined disease proneness of plants is fraught with great methodological difficulties.

Furthermore, the extent of disease proneness can be established only subsequently from the type of the infection and the course of the disease, whereas the thermally determined behaviour of the host can only be studied independently of that of the parasite (p. 229) in the pre-infectious period, since, after infection has occurred, both host and parasite are exposed together to the same environmental temperature.

In addition, under natural conditions, change in temperature is the rule, and the rhythm and range of these changes are characteristic. For the present, in order to gain a first insight into the problem, we work in the laboratory at constant temperatures, i.e. under artificial conditions.

In order to simplify the discussion we shall again divide the thermally modified disease resistance of the host into its four components: (a) resistance to attack, (b) resistance to penetration, (c) resistance to spread,

and (d) reactivity. It will be clear, of course, that the outer temperature does not influence these four aspects consecutively, all are affected simultaneously and sometimes in opposing directions; it is solely for reasons of economy that we shall now discuss one aspect at a time, as if it were the most important, and neglect the others without continually repeating this caveat.

(a) *The Influence of Environmental Temperature on Resistance to Attack*

As a rule, the environmental temperature affects resistance to attack by lengthening or shortening the period of development in which susceptible individuals are open to infection. For this reason, lower temperatures

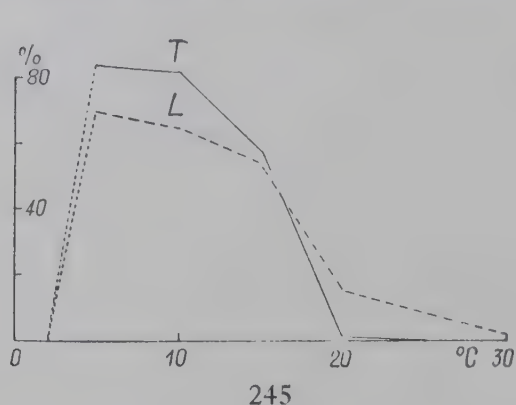


FIG. 245. The influence of soil temperature on bunt attack of wheat, var. Dawson. Abscissae: soil temperature. Ordinates: number of diseased individuals. Curve L: *Tilletia laevis*. Curve T: *Tilletia tritici*. (After Faris, 1924.)

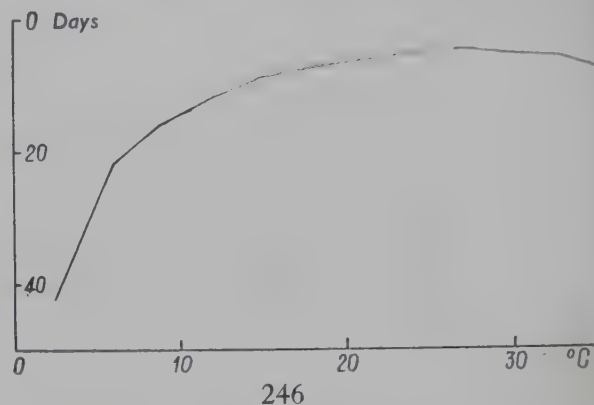


FIG. 246. The influence of germination temperature on germination rate of Plantahof wheat. Abscissae: germination temperature. Ordinates: number of days required by seedlings to reach a length of 5 cm., counted from the moment of moistening the grain, i.e. the time needed to reach the soil surface. (After Gäumann, 1932.)

normally tend to depress resistance to attack. In a particular experiment (Fig. 245) the optimum temperature for attack by bunt of wheat was 5° C., which is much below the optimum both for germination of brandspores (p. 29) and for the development of wheat seedlings (Fig. 246). This optimum is explained by the fact that at 5° C. the conditions for the meeting of parasite and host are most favourable; this temperature not being so low as to prevent sufficiently large numbers of brandspores from germinating in a reasonably short time, but being low enough to keep the wheat seedlings in their susceptible stage of growth for a long time. In a different variety of wheat (Fig. 246) it has been shown that at 27° C. the plant needs on an average about 5 days to reach a height of 5 cm., but at 5° C. at least 27 days; a similar shift probably also occurs in the time of exposure to infection, and thus the chance of successful infection must vary with the prevailing temperature.

At 5° C., however, the lower temperature limit for bunt infection is nearly reached; the temperature optimum for infection thus lies immediately above the temperature minimum. If now another factor of the environment (e.g. soil moisture) be sub-optimal, then the development of

the parasite is immediately retarded and the incidence of attack falls in spite of the long duration of the susceptible stage in the host's development. The extent of attack, therefore, cannot be calculated as the arithmetic mean of the vital requirements of the host and parasite, since it is just in these threshold regions that it is very labile, uncertain, and a law unto itself.

The effect of time of sowing on attack by bunt of wheat is, therefore, partly to be explained by the influence of external temperature on resistance to infection (Fig. 247).

The thermally determined infection axeny in onion smut (*Urocystis cepulae*) is as striking as that in bunt of wheat. Here, environmental

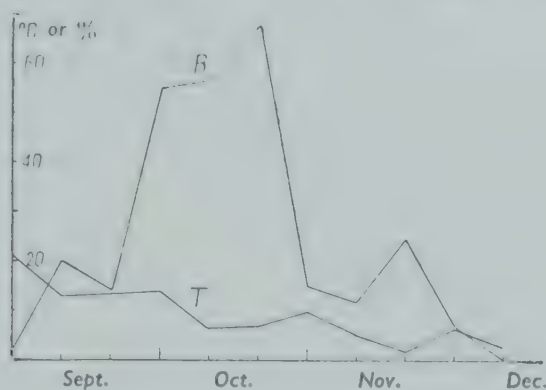


FIG. 247. The effect of sowing time on bunt attack of winter wheat. Abscissae: date of sowing. Curve *T*: meteorological 5-day means of temperature. Curve *B*: bunt attack. (After Hahne, 1925.)

conditions can push the developmental rhythms of host and parasite so far apart that they become the limiting factor determining the geographical distribution of the disease.

As soon as the onion seed germinates the seedling can be infected—with greatest certainty during the first 5 days. Hardly any infection occurs after the production of the first leaf and, therefore, soil temperatures during the short critical period are decisive.

Below 10° C. the development of the host is retarded and the growth stage during which it may be infected is prolonged. The pathogen, of course, is also retarded, but this does not prevent germination of its brandspores and the 'taking' of infection, only its further progress. As soon as the days get warmer the pathogen can resume its growth in the tissues; hence, the incidence of infection is correspondingly high below 10° C.

At temperatures between 10° and 25° C., host and parasite are equally favoured though the incidence of attack is somewhat reduced.

At 25° C. the host is stimulated to even more rapid development, but the pathogen is adversely affected and tends to fall behind. Even at 27° C. the disease incidence falls rapidly, and at 29° C. infection no longer occurs (Walker and Wellman, 1926).

On account of this micro-climatically determined separation of their developmental rhythms, the parasite is practically unable to attack the

host in warm soil. The onion smut is, therefore, confined to the cooler northern regions of Europe and North America; it must constantly be reintroduced to the warmer regions in the course of world trade but inevitably perishes.

(b) *The Influence of Environmental Temperature on Resistance to Penetration*

On page 336, in our discussion of *Thielavia* rot of tobacco, we were dealing with a disease in which soil temperature profoundly modifies resistance to penetration. In susceptible varieties of tobacco, at low soil temperatures, the formation of root periderm is considerably delayed so

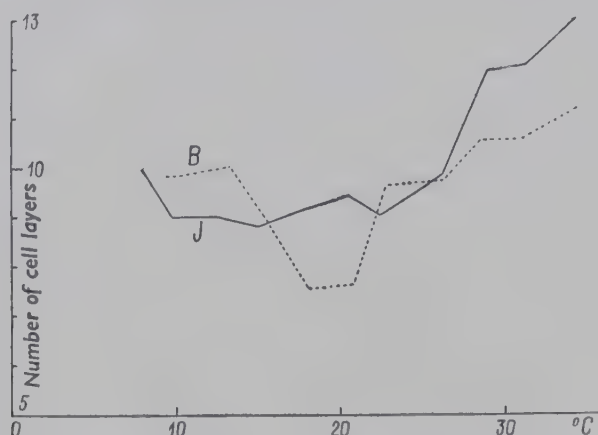


FIG. 248. The influence of soil temperature on the number of cell layers in the periderm of the potato, var. Bintje (curve *B*) and var. Ideal (curve *J*). (After Gäumann and Häfliger, 1944.)

that the roots are accessible to infection for a long time, whereas at high temperatures periderm is formed early and the protective mantle is soon completed.

A similar influence of soil temperature on the structure of peripheral layers, and hence on resistance to penetration, exists in tubers, rhizomes, &c., although here conditions are much less clear; potato tubers, for instance, produce the thinnest skin at medium and not at the highest temperatures (18–21° C., Fig. 248).

Sometimes environmental temperature may influence resistance to penetration by affecting the rate of healing of wounds caused by snails, rodents, &c., in the soil, since the more rapidly these wounds are closed the shorter is the exposure to infection by wound parasites. In gladiolus corms neither cork nor wound periderm formation takes place at 0° C.; the former begins at 4.5° C. and reaches a maximum at 21.9° C., whilst the latter only starts at 15.3° C. and reaches its maximum at 30.9° C. (Fig. 249).

(c) *The Influence of Environmental Temperature on Resistance to Spread*

It is probable that environmental temperature affects resistance to spread through its influence on the differential formation of cell walls, e.g. in maturing shoots of fruit trees or of forest trees near the tree line. It may

be assumed that poorly matured shoots, whose lignins, for example, are still insufficiently methoxylated, possess less resistance to the spread of parasites (they are also more susceptible to frost damage), although these relations have not yet been determined quantitatively.

Rather more thorough investigations have been carried out on the susceptibility of cereal seedlings. The pathogens of seedling diseases can readily break down hemicelluloses and protopectins, but not cellulose or lignins. Hence, one would expect that the variation in the content of the former substances in the cell walls, as determined by the growth temperature, would constitute an important factor in resistance to spread.

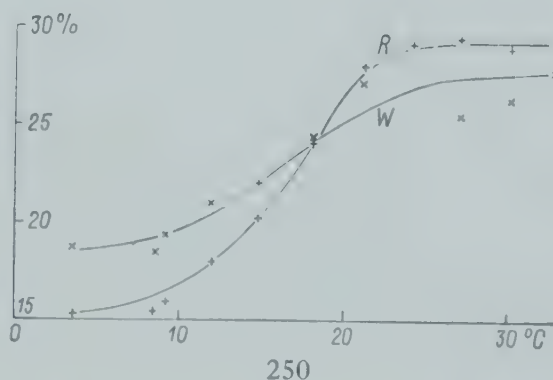
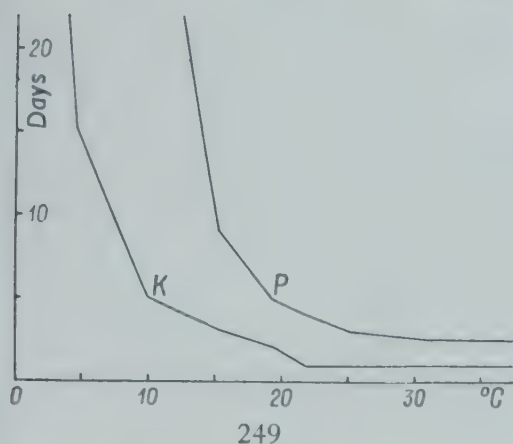


FIG. 249. The influence of temperature on the rate of cork formation (curve *K*) and wound periderm formation (curve *P*) in gladioli. Ordinates: days after cutting. Abscissae: temperature. (Schematized after Artschwager and Starrett, 1931.)

FIG. 250. Solubility by the enzymes of *Fusarium herbarum* of cell wall materials of cereal seedlings grown at different temperatures. Curve *R*: rye seedlings (Swiss Landroggen). Curve *W*: wheat seedlings (Plantahof wheat). Abscissae: growth temperature of seedlings. Ordinates: percentage solution by enzyme mixture. (*W* after Gäumann, 1932; *R* original.)

In fact, there is a direct correlation between the temperature at which the seedlings are raised and the solubility of their cell walls by fungal enzymes. Fig. 250 shows that, *ceteris paribus*, the cell walls of wheat seedlings raised at 33° C. are approximately 50% more soluble than those of seedlings raised at 3° C., and in rye seedlings the solubility is almost doubled. Thus, the consistency of cell walls decreases with the rate of shooting of the seedlings (Fig. 246). The causes of this increase in solubility lie partly in differences in the chemical composition of the cell walls (Table LXII; Fig. 251), and partly in their differential sub-microscopic structure.

In some seedling diseases an increase in susceptibility is found actually accompanying the greater solubility of the walls. For instance, the temperature curves of the *Fusarium* diseases of rye (curves *N* and *H* in Fig. 252) and of wheat (curves *G* and *C* in Fig. 253) rise on the whole concurrently with the solubility of the cell walls (Fig. 250) and appear to be unrelated to the glucose and saccharose contents of the seedlings (Table LXII). Admittedly, the disease curves differ from one another and from the cell wall curves in many details but, in any case, the correlation with

the curve for cell wall solubility is much closer than, for instance, with the curves of the thermally conditioned growth-rate of the pathogen (Fig. 254). The latter are saddle-shaped and tend to fall again at high temperatures, whereas the disease curves reach a definite climax with high soil temperatures. Thus, it is certainly not the thermally conditioned vitality of the pathogen which, in this instance, determines the shape of the curve but the thermally conditioned decrease in the resistance to spread and in

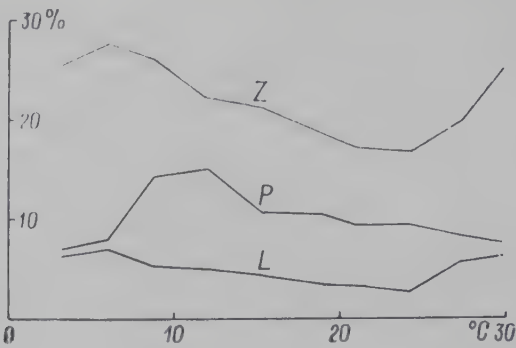


FIG. 251. The influence of germination temperature on the chemical composition of the cell wall of rye seedlings (Swiss Landroggen). Abscissae: temperature during growth. Curve P: protopectins. Curve Z: cellulose. Curve L: lignins as percentage of total wall materials. Original.

the vitality of the host. As the growth temperature rises, the cell walls become increasingly soft and more readily soluble, the whole seedling organism expends itself in growth in length and becomes ever less resistant. The same is true of the damping-off of spruce (*Picea excelsa*) caused by *Pythium de Baryanum* and *Fusarium bulbigenum* (Roth, 1935).

TABLE LXII

Influence of growth temperature on the chemical composition of rye seedlings (Swiss Landroggen; contents as % dry weight.) Original

Growth temperature	Glucose	Saccharose	Dextrine	Starch	Total carbohydrate	Protopectins	Hemicellulose A	Hemicellulose B	Total hemicellulose	Cellulose	Lignins	Sum of cell wall materials
° C.	%	%	%	%	%	%	%	%	%	%	%	%
3.2	8.8	3.4	0.4	0.8	13.4	3.4	19.4	9.8	32.6	12.2	3.2	48.0
6.0	8.3	6.2	0.4	1.2	16.1	3.6	15.2	9.8	28.6	12.0	3.0	43.6
8.9	9.9	2.9	0.3	1.0	14.1	5.9	10.0	12.3	28.2	10.7	2.1	41.0
12.0	12.2	4.4	0.1	1.1	17.8	6.5	8.9	16.5	31.9	9.6	2.2	43.7
15.4	12.6	4.4	0.2	1.0	18.2	4.2	11.8	13.4	29.4	8.4	1.8	39.6
18.9	11.0	1.3	0.2	1.0	13.5	4.8	14.3	16.5	35.6	8.4	1.6	45.6
21.0	11.4	3.7	0.1	2.0	17.2	4.5	20.7	12.6	37.8	8.0	1.5	47.3
24.2	8.9	5.7	0.2	1.5	16.3	4.1	17.1	13.5	34.7	7.2	1.2	43.1
27.1	7.8	5.9	0.4	1.6	15.7	3.7	18.5	11.7	33.9	8.9	2.6	45.4
29.9	8.9	5.7	0.3	2.1	17.0	3.6	19.1	10.8	33.5	12.1	3.0	48.6

In other seedling diseases, such as the *Fusarium* diseases of rice, e.g. that caused by *Fusarium herbarum* (Fig. 252, curve *R*), it is not the thermally conditioned susceptibility of the host which is dominant but the thermally increased vitality of the parasite. In spite of an increasing solubility of the cell walls, the curves for disease incidence approximate closely to those of the growth-rates of the parasites. This contrast between

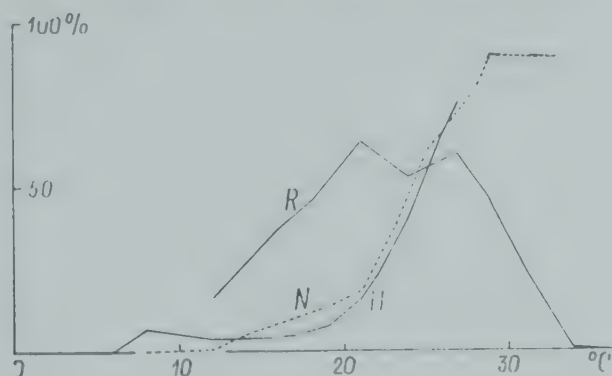


FIG. 252. The influence of soil temperature on the incidence of *Fusarium* disease of rye and rice seedlings. Curve *N*: Petkus rye with *Fusarium nivale*. Curve *H*: Swiss landroggen with *Fusarium herbarum*. Curve *R*: Swiss Landjah rice with *Fusarium herbarum*. Abscissae: soil temperature. Ordinates: number of diseased plants as percentage of surfaced plants. (After Anliker, 1935, and de Haan, 1937.)

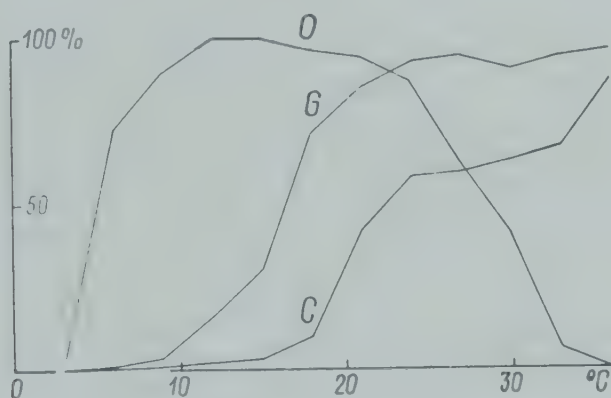


FIG. 253. The influence of soil temperature on the occurrence of seedling diseases in wheat. Curve *G*: *Gibberella Saubinetii*. Curve *C*: *Fusarium culmorum*. Curve *O*: *Ophiobolus graminis*. Abscissae: soil temperature. Ordinates: number of diseased plants as percentage of surfaced plants. (After Tanja, 1933, and Krebs, 1933.)

the dominance of host and pathogen is again seen in curves *H* and *R* of Fig. 252, where the same pathogen is used but with different hosts.

In still other instances, e.g. take-all and whiteheads of wheat (*Ophiobolus graminis*), the shape of the curve of the temperature-controlled disease incidence (Fig. 253, curve *O*) is determined mainly by the thermally conditioned vitality of the pathogen (Fig. 254, curve *O*), much more so in any event than by the differential solubility of the cell walls of the host (Fig. 250). In spite of this, the thermal optimum for disease incidence (12–15° C.) is markedly sub-optimal for the pathogen. Other determining factors, therefore, must be involved, chiefly the impaired reactivity of the

host at low temperatures. This factor will be considered further under (d) and again in sub-section 7.

(d) *The Influence of Environmental Temperature on the Reactivity of the Host*

A direct influence of environmental temperature on the capacity of the host to react pathologically may be surmised in those cases where the

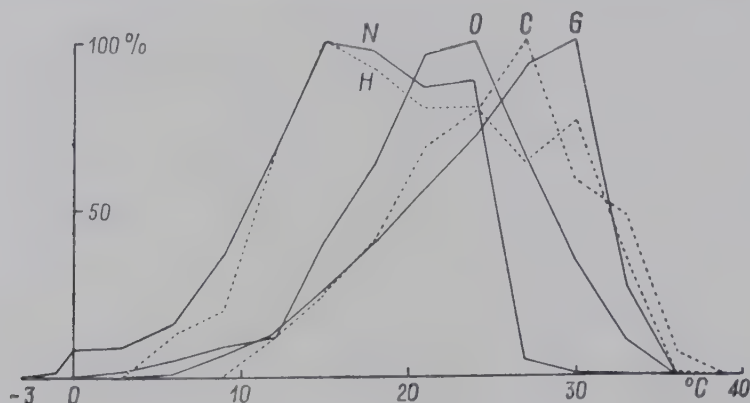


FIG. 254. The influence of temperature on the growth-rate of some pathogens of seedling diseases of cereals. The maximal growth-rate as determined from mycelial dry weights was assigned the value of 100 in each case. Curve H: *Fusarium herbarum*. Curve N: *Fusarium nivale*. Curve O: *Ophiobolus graminis*. Curve C: *Fusarium culmorum*. Curve G: *Gibberella Saubinetii*. (After Tanja, 1933, and Anliker, 1935; C original.)



FIG. 255. The influence of soil temperature on the average yield of tubers per plant in the potato, var. Bintje. (After Gäumann and Häfliger, 1944.)

thermally governed disease curve differs from the temperature curve of the pathogen. There is a large number of infectious plant diseases in this category (Fuchs, 1933) and we shall select two examples, one an infectious disease of potato tubers and the other of cereals.

The metabolism of potato tubers is surprisingly responsive to soil temperature, as is shown by the graph in Fig. 255. Although the foliage develops about equally well over a wide range of soil temperatures, yet below 13°C. and above 26°C. only a small part of the assimilate is stored in the tubers and, in consequence, outside these limits the yield of tubers falls steeply. In spite of an equal assimilatory capacity of the foliage, the

economic yield of tubers is small outside the temperature range mentioned, since the assimilate is evidently respired away again immediately by the subterranean organs.

It was seen earlier from Fig. 248 that the skin of the potato tuber varies in its structure according to conditions during its growth. Fig. 256 demonstrates further that the starch-free surface layer of the primary

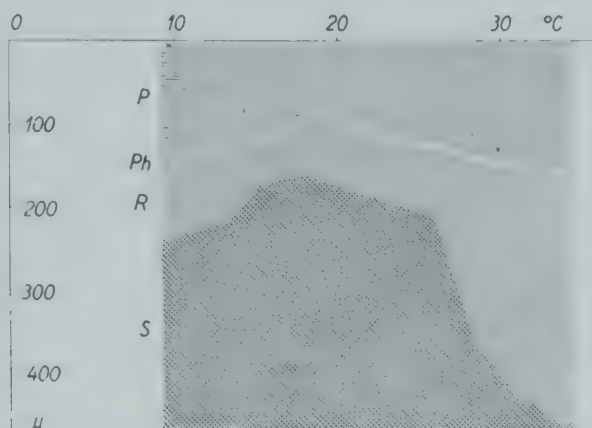


FIG. 256. The influence of soil temperature on the composition of the peripheral layers of tubers of the potato, var. Bintje. *P* periderm; *Ph* phellogen; *R* starch-free and *S* starch-containing primary cortex. (After Gäumann and Häfliger, 1944.)

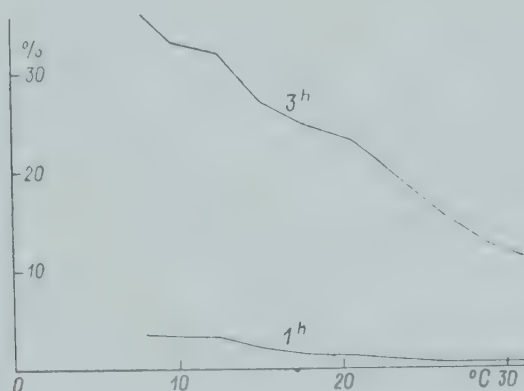


FIG. 257. The influence of soil temperature on the resistance of starch to hydrolysis by hydrochloric acid, i.e. its solubility in 1 or 3 hours respectively (var. Ideal). (After Gäumann and Häfliger, 1944.)

cortex remains of normal thickness (about $50\text{--}90\mu$) at temperatures up to about 26°C ., but above this the thickness increases rapidly, and at 34°C ., under certain circumstances, it may constitute the whole tuber; for example, in the variety Ideal such tubers are practically starch free. Finally the quality of the starch, especially its sub-microscopic structure, is decisively affected by the temperature at which the tubers are grown (Fig. 257).

Corresponding with these changes in physiological condition, the reactivity of potato tubers must also vary with different external temperatures. Their behaviour towards an ectoparasite (*Actinomyces scabies*) and an endoparasite (*Phytophthora infestans*) may be cited in illustration.

Actinomyces scabies (common scab of potato) invades the growing tuber through its lenticels (Fig. 50) and through wounds. The minimum temperature for its growth is 3°C ., the optimum 24°C ., and the maximum 33°C . (Fig. 258). However, attack by common scab begins only at a soil temperature of about 10°C . (Fig. 259), reaches its maximum at about $13\text{--}15^{\circ}\text{C}$., and then slowly declines. If the soil temperature be higher than 26°C . there is practically no scab attack. The disease optimum lies, therefore, at a soil temperature which is sub-optimal for the pathogen (Fig. 258), whereas it corresponds closely with the thermally conditioned yield optimum of the host (Fig. 255).

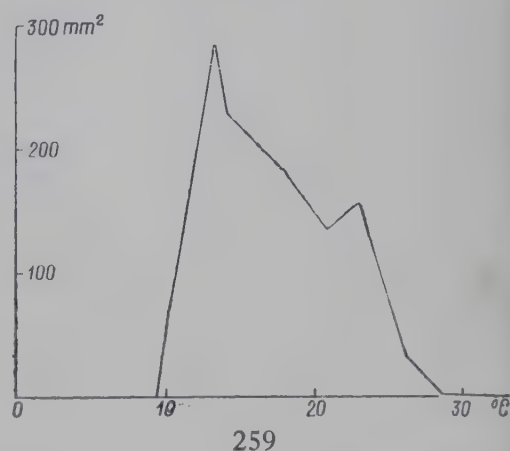
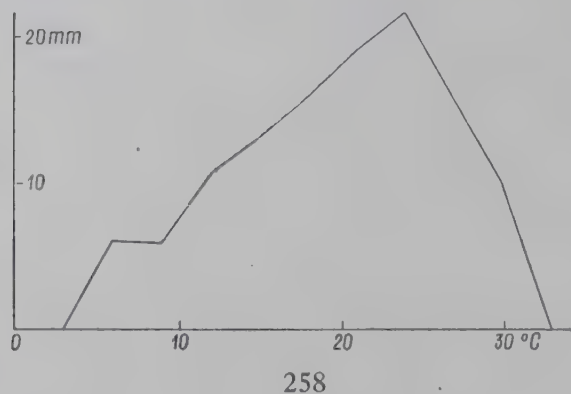


FIG. 258. The influence of temperature on the growth-rate of a strain of *Actinomyces scabies*. Ordinates: diameter of the cultures after 117 days. (After Gäumann and Häfliger, 1944.)

FIG. 259. The influence of soil temperature on the incidence of common scab on potato tubers. Abscissae: soil temperature. Ordinates: amount of scabbed surface per plant; sum of three experiments. (After Gäumann and Häfliger, 1944.)

We may surmise that infection through the lenticels of young tubers at temperatures optimal for the pathogen occurs at least as frequently, if not more frequently, as under sub-optimal conditions. Hence, if the disease develops only in the latter case, this must be due to a difference in the reaction proneness of the host. The more vigorous the host, the greater its readiness to react pathologically (p. 382), and the greater the incidence of the skin disease termed common scab.

It is still unknown why infections cease to develop above 26°C .; either the host is tolerant so that the infections fade out without symptoms, or alternatively, it shows necrogenous hypersensitive reactions so that the pathogen at once becomes encapsulated in the lenticels without inflicting macroscopic damage. Whatever the reason, it is rather striking that the temperature limit of 26°C . coincides with the turning-point between storage and non-storage of starch in the primary cortex (Fig. 256).

The thermally determined reactivity of the potato to *Phytophthora infestans* (potato late blight; Fig. 271) manifests itself in an entirely different way. Because the influence of temperature on the defence capacity of the tissues is of major practical interest in stored potatoes, in the present experiments it is not the temperature at which the tubers were grown that

has been varied but the storage temperature (of tubers all grown under the same conditions).

Fig. 196 shows that the temperature minimum for the pathogen is 4°C ., the optimum $19\text{--}20^{\circ}\text{C}$., and the maximum only $25\text{--}26^{\circ}\text{C}$.

The defence reactions of potato tissues to *Phytophthora infestans* were discussed on page 298, together with their temperature sensitivity; Fig. 260 represents the latter graphically. Below 10°C . the proneness of the cells to necrogenous aborting reactions increases with rising temperature, whereas over 12°C . it is little influenced by the external temperature. At low

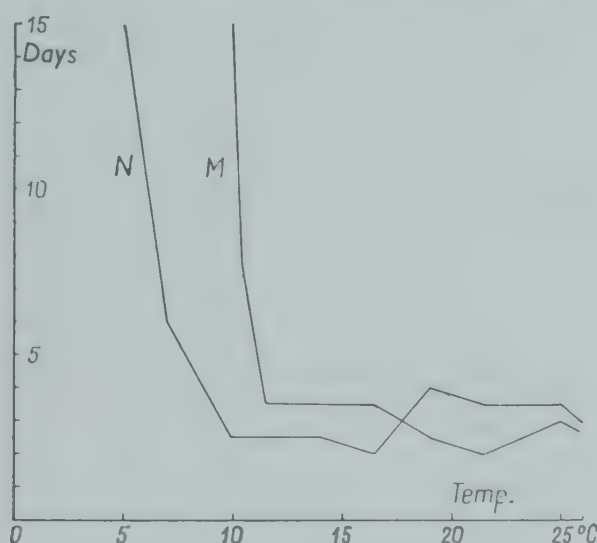


FIG. 260. The influence of temperature on the reaction of potato tissues (var. Erdgold) to *Phytophthora infestans* (dry rot of potatoes). Curve *N*: beginning of necrosis. Curve *M*: beginning of mycelial development inside the tuber tissues. (After Müller and Griesinger, 1942.)

temperatures, however, the vitality of the parasite is impaired to a greater extent than is the reaction proneness of the host. The parasite develops slowly and probably also rather weakly; it is unable to outgrow the necrogenous reactions of the host and is killed by them. Hence, under 10°C . it does not succeed in forming reproductive bodies on the tubers, i.e. it does not propagate itself and therefore the stored potatoes remain healthy.

Late blight resembles common scab in that the thermally conditioned vitality of the pathogen (Fig. 196) in no way determines the disease curve (Fig. 260, curve *N*). In contrast to potato scab, however, the disease curve is not determined by the thermally conditioned vitality of the host but rather by an interplay between pathogen and host whose end effect cannot be predicted quantitatively. The host, with its necrogenous defence reactions, must injure itself in order to keep the pathogen at bay and, therefore, the maximum defence success occurs where the host is near and the pathogen right at the lower limit of their powers, namely, at a temperature of $4\text{--}5^{\circ}\text{C}$.; hence the success of cool storage for potatoes.

Extraordinarily complex relations, equalling those of mineral nutrition (p. 388), obtain in the second example, the temperature-induced change of

reaction norm in infectious diseases of cereals, in particular, of rust diseases. The more finely the pathogen-host relationship is differentiated, the more sensitively it responds to environmental influences. As in the case of mineral nutrition, the external temperature only modifies the reactivity in intermediate pathogen-host pairs, but not in those at either extreme. For instance, Table LXIII shows that the highly resistant spring wheat variety, Rümkers Sommerdickkopf, remains highly resistant at almost all external temperatures, and the highly susceptible winter variety, Strubes Winterdickkopf, remains highly susceptible. Again, Egyptian barley (Table LXIV) is very resistant to biotype 19 of the brown rust at all temperatures, whereas mountain barley is highly susceptible to the same biotype at all temperatures.

TABLE LXIII

Influence of temperature on the susceptibility of some wheat varieties to Puccinia glumarum tritici, biotype 4. (After Gassner and Straib, 1929)

Variety	Temperature	Percentage of plants with infection type					
		i	o	I	2	3	4
	° C.	%	%	%	%	%	%
Rümkers Sommerdickkopf wheat . . .	10-11	52.7	18.5	0	0	28.8	0
	15	96.2	0	0	0	3.8	0
	19-20	96.3	2.2	0	0	1.5	0
Svalöfs Panzer wheat III	10-11	0	0	0	100	0	0
	15	13.4	0	0	86.6	0	0
	19-20	82.4	9.1	0	8.4	0	0
Aurore summer wheat . . .	10-11	0	13.7	12.3	0	68.5	5.5
	15	4.6	10.6	0	0	83.4	1.3
	19	44.7	9.9	0	0	45.4	0
Strubes Dickkopf winter wheat	10-11	0	0	0	0	0	100
	15	0	0	0	0	0	100
	19	0	0	0	0	0	100

In intermediate types the environmental temperature modifies the speed of reaction, the type of reaction, and the direction of change of reaction.

With rising temperatures the change of speed of reaction shows itself mainly in a reduction of the incubation period and of the time to fructification; at low temperatures rust infection proceeds more slowly, at higher temperatures more quickly.

Examples of modification of the type of reaction can be found in Tables LXIII and LXIV. At temperatures of 10-11° C., Svalöfs Panzer wheat (Table LXIII) shows 100% infection of medium severity, but at 19-20° C. it is practically immune. The answer to the question whether or not a variety is susceptible to rust will differ according to the prevailing temperature. Thus, in cool weather (or if cultivated in a corresponding

climatic region) Svalöfs Panzer wheat would be called medium susceptible, but in warm weather (or warm regions of cultivation) it is highly resistant. Hence, a shift of a mere 10° C. in the temperature, such as occurs in the normal alternation of day and night, may, under certain circumstances, show the rust resistance of a given variety in quite a different light.

TABLE LXIV

*Influence of temperature on the mean susceptibility of some barley varieties to several biotypes of Puccinia hordei (barley brown rust).
(After Straib, 1936)*

Barley variety	Temperature ° C.	Biotype 1	Biotype 10	Biotype 16	Biotype 19
Berg	11.9	00	00-i	4	4
	16.1	0	0	4	4
	21.5	1	1	4	4
Egyptian	11.9	0	4	4-3	0
	16.1	0-1	4-3	4-3	0
	21.5	0	4	3-4	0
Cruzat	11.9	0	0	3-4	0
	16.1	2	0	4	2
	21.6	3-2	2-3	3-4	2-3

The critical temperature at which the reaction norm changes is specific for each variety; Table LXIII shows that it lies between 10° and 15° C. for Rümkers Sommerdickkopf wheat, and between 15° and 20° C. for Svalöfs Panzer wheat. Above 20° C. the results of infection trials with rust fungi are almost always unreliable.

In addition to the extent, the direction of the temperature-induced change of reactivity differs according to the parasite-host pair concerned (as in the influence of mineral nutrition). In general, the resistance of cereals to black rust, *Puccinia graminis*, diminishes with rising temperatures (Fig. 261), whereas resistance to yellow rust, *P. glumarum* (and less markedly to brown rust, *P. triticina*), increases with rising temperature. The two rusts thus show, in their temperature reaction, the same contrast as in their developmentally conditioned changes of immunity level: youth resistance-age susceptibility in many black rust biotypes, youth susceptibility age resistance in certain yellow rust biotypes (p. 376).

As in the case of nutrition, all statements about general changes of reactivity due to the influence of temperature must be regarded only as rough generalizations: they are valid only for a certain number of biotypes and not for others. An example given in Table LXIV is the barley variety Cruzat, which is highly susceptible at all experimental temperatures to biotype 16 of the barley brown rust, *P. hordei* (= *P. simplex* = *P. anomala*), whereas its reaction to biotypes 1 and 10 is very sensitive to temperature.

In finely differentiated pathogen-host pairs each combination of a given host variety with a given strain of pathogen responds characteristically to the environmental temperature: there is no such thing as a general optimal temperature for infection, equally valid for all strains of pathogen and all varieties of host.

Confirmation of these results is given in Tables LXV and LXVI for the leaf stripe disease of barley. The temperature minimum for the pathogen is about 0°C ., the optimum about 25°C ., and the maximum about 33°C . The establishment and course of the parasitic relationship

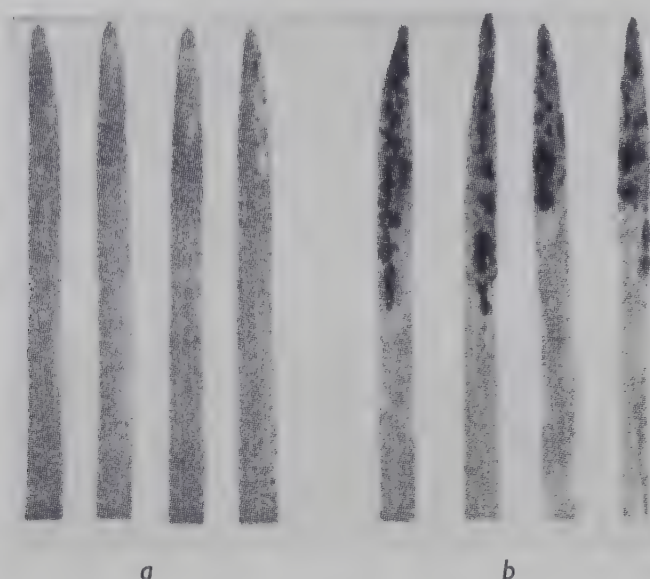


FIG. 261. The influence of air temperature on the reaction of seedlings of *Triticum spelta* (spelt wheat, var. März) to biotype 29 of *Puccinia graminis tritici* (black rust of wheat). *a* air temperature 14.4°C . *b* 24.0°C . (After Newton and Johnson, 1932.)

are greatly influenced by temperature, but its effects differ according to the variety and biotype, and may even contrast with one another. It is not simply a race between pathogen and host, e.g. an outgrowing of the pathogen by the host or a general weakening of the host: the outcome, the number of infected individuals that later become diseased at the different growth temperatures, will evidently be decided by the total complex which we summarize as the reactivity of the host and as the mutual conflict between pathogen and host.

These brief notes do not, of course, by any means exhaust the possibilities of temperature-conditioned change in rust susceptibility. Since the reaction norm of an individual host may change during the course of ontogenetic development as well as under the influence of external temperature, it is practically certain that, on occasion, the temperature-conditioned reaction norm of an individual host may also change in the course of development (Straib, 1940). However, the fact that emerges sufficiently clearly from the above examples and is of interest to us here is that, in labile forms, the infection type is not a varietal characteristic, it is not constant and final but plastic. It is not a definite condition,

a given level of immunity, that is characteristic of such a variety and specific to it, but the proneness to variation and the range of variation of the defence reactions.

TABLE LXV

*Influence of infection temperature on attack of some barley varieties by three biotypes of Helminthosporium gramineum (leaf stripe).
(After Isenbeck, 1937)*

Variety	Biotype	Percentage attack at a temperature of				
		5° C.	10° C.	15° C.	20° C.	25° C.
Velvet	3501-11	% 79.3	% 93.1	% 65.5	% 24.5	% 30.6
	68-1	78.7	93.6	63.6	65.6	54.4
	118-2	9.6	5.1	4.7	0	0
Morgenrot	3501-11	58.8	39.5	7.3	5.0	12.9
	68-1	18.9	36.6	10.2	6.0	2.7
	118-2	9.4	2.8	0	0	1.3
Heines Hanna	3501-11	68.4	81.1	52.7	42.3	66.2
	68-1	12.0	32.8	14.7	8.4	2.6
	118-2	29.0	13.1	14.8	5.2	1.5
Heines four-rowed. . . .	3501-11	41.0	59.6	14.2	0	4.2
	68-1	44.9	26.5	55.8	20.0	18.3
	118-2	82.7	79.5	46.9	13.2	5.4

TABLE LXVI

Influence of different infection and growth temperatures on the percentage attack of Heines Hanna barley by Helminthosporium gramineum (leaf stripe). (After Isenbeck, 1937)

Biotype	Infection temperature 10° C.; subsequent growth temperature		Infection temperature 20° C.; subsequent growth temperature	
	10° C.	15-22° C.	10° C.	15-22° C.
3501-11	% 4.7	% 40.9	% 25.0	% 7.6
59-3	0	13.8	0	5.2
68-1	4.7	15.6	5.2	0

Moreover, in the field, the reaction type will be influenced not only by the air temperature but, at the same time, also by the stage of development, nutrition, water-supply, assimilation, &c., all factors which in part reinforce and in part oppose one another. Admittedly, all are not of equal importance, but if one were to consider the effects of only one pair of factors, e.g. 'varying nitrogen supply' versus 'varying temperature', for the different pathogen-host combinations, one would require several reams of paper.

In view of this interplay of different kinds of factors, it is easy to appreciate the difficulties standing in the way of an interpretation of field

trials on susceptibility or resistance to disease under natural conditions. A mixture of several biotypes will almost certainly be present, which may react variously to given environmental factors; further, the several factors will not act in a constant manner on the course of the parasitic relationship, but will vary according to weather and time of day.

Of course, among this multitude of individual factors there will be some prime factors (e.g. temperature) that will dominate the lesser factors so that, sometimes, certain regularities appear in the final issue. In black rust, for instance, the phase-specific increase of susceptibility during the growing period (p. 375) combines with the heightening of susceptibility due to rising temperature (p. 405) to produce a summation effect; hence, outbreaks of black rust tend to be particularly severe in warm seasons and in warm climates.

In contrast, in the climatic conditions of central Europe, attack by yellow rust generally reaches a maximum during the cool months of spring and early summer, receding with the advent of warmer summer temperatures: the outbreak of uredo pustules is now replaced by necrotic discolorations and, in consequence, fresh infections either do not occur or occur only sparsely. The plants, therefore, show a midsummer lessening of their rust infection which is known in practice as field resistance or summer resistance. It is caused, partly, by a temperature-induced rise in resistance of the host and, partly, by a temperature-induced reduction of the vitality of the pathogen (Küderling, 1936).

5. *The Influence of Humidity on the Disease Proneness of the Host*

Only rarely do the moisture relations of the environment exert any direct influence on the disposition of man to infectious diseases (the disposition to rheumatic diseases in humid climates is an exception), but, in some cases, they are able to modify the disease proneness of plants very considerably. The influence may be exerted by way of soil moisture, atmospheric humidity, or by a modification of the water economy of the host. The influence of water relations on the occurrence of infection itself will not be discussed here (see p. 26).

(a) *The Influence of Soil Moisture on the Disease Proneness of the Host*

In the simplest case, e.g. in the smut diseases of cereals, soil moisture alters only the disposition to infection (incidence of attack) and not the disposition to disease (course of disease). For example, low soil moisture increases the incidence of attack in loose smut of oats (*Ustilago avenae*) and in millet smut (*Sorosporium Reilianum*) (Table LXVII).

One might expect the incidence of disease to be greater in moist than in dry soils, as the former are more favourable to the germination of the brandspores of the parasite and their further budding. But, in spite of better conditions for the development of the pathogen, the incidence of disease is only one-half as great in moist as in somewhat drier soils, because higher soil moisture favours the development and vitality of the

host even more than the pathogen; the seedlings develop faster and out-grow the pathogen, the higher moisture having produced increased resistance to attack, and perhaps also enhanced their defensive powers.

Table LXVII shows interference between two factors of different types. At the optimal soil moisture for smut attack (15% of the water-holding capacity) soil moisture prevails over soil temperature and thus, even at unfavourable temperatures (below 20° C.), a considerable number of individuals still become diseased. However, if two factors unfavourable to smut attack occur together, low soil temperature and increased soil moisture (25%), their effects are summated and attack does not take place.

TABLE LXVII

Influence of soil moisture and soil temperature on disposition to infection by the millet smut. (After Christensen, 1926)

Soil temperature ° C.	Percentage attack at a soil water content of	
	15%	25%
	%	%
12-15	2.8	0
16	16.2	0
20	23.9	4.5
24	26.5	5.2
28	46.5	23.4
32	19.2	13.3
36	10.9	0.5

In most other infectious diseases of plants which are responsive to soil moisture, the latter changes not only the disposition to infection but also that to disease.

In one group, disposition to disease heightens with increasing soil moisture. In some of the diseases, e.g. soft rot of potato, any such heightening of disease proneness is directly reflected in the yield. In the experiment shown in Table LXVIII, healthy, uninjured potato tubers were planted in soil contaminated with *Bacillus atrosepticus*. Five levels of soil water content were employed varying from 25% to 125% of the water-holding capacity of the soil. At low soil temperatures (6-10° C.), natural infection of tubers took place only if the soil moisture exceeded 100% but, at soil temperatures of 15° and 20° C., infection was 100% successful even where the soil was only just saturated with water. With lower water contents the percentage infection fell rapidly and at a water content under 50% all tubers remained healthy. In addition to the disease incidence (the number of spontaneously infected tubers) the disease itself (the 'rot', i.e. the relative proportion of destroyed tissues) was greatly influenced by the soil moisture.

In some other infectious diseases, however, the correlation between increased soil moisture and heightened disease-proneness is not yet so clear. The foot rot of beans (*Fusarium Martii* f. *phaseoli*) attacks the plants

more severely when the soil moisture is high than when it is low; on the other hand, the plants are able to produce replacement roots more freely, thus balancing the increased attack. Therefore, if yield were adopted as a measure of the degree of disease, it would be concluded (wrongly) that the plants are more susceptible at a low soil moisture (25% of the water-holding capacity) because the loss is greater than at a high soil moisture content (45 and 60%; Burkholder, 1924). In the same way, the heavier incidence of take-all of wheat (*Ophiobolus graminis*) under high soil moisture conditions is compensated by the formation of replacement roots (McKinney and Davis, 1925).

TABLE LXVIII

Influence of soil moisture and soil temperature on the occurrence of wet rot of potato tubers, variety Modrows Industrie. (After Fehmi, 1933)

Water content	Soil temperature					
	6-10° C.		15° C.		20° C.	
	Diseased tubers	Diseased tissues	Diseased tubers	Diseased tissues	Diseased tubers	Diseased tissues
%	%	%	%	%	%	%
25	0	0	0	0	0	0
50	0	0	10	1	20	1
75	0	0	50	20	60	23
100	0	0	100	60	100	73
125	100	50	100	40	100	73

In a second group of infectious diseases, the graph relating soil moisture to susceptibility shows two limbs; these may be either concave or convex relative to the x -axis.

The first case, little disease at lower and higher levels of soil moisture, and severe disease at medium levels, is exemplified by the *Fusarium* disease of wheat seedlings (*Gibberella Saubinetii*) and the wilt disease of tomatoes (*Fusarium lycopersici*). In one particular experiment (Dickson, 1923) the incidence of *Fusarium* disease of wheat was 60% at a soil moisture of 30% (relative to the water-holding capacity), 84% at 45% water content, and 48% at 60% water content. Hence, medium soil moistures favour attack of wheat by *Gibberella*, and high soil moistures favour attack by *Ophiobolus*. This statement applies, however, only at the optimum soil temperature for *Gibberella*, 23° C. (Fig. 253, curve G). At a soil temperature of 8° C. and an average moisture of 30%, fully 72% of the wheat seedlings were attacked, 44% at a water content of 45%, and 0% at a water content of 60%. The effects of soil temperature and soil moisture may, therefore, either reinforce or counteract one another. The curves of Fig. 252, &c., are valid only for a given soil moisture.

The effect of soil moisture on disposition to wilt diseases is as variable as on that to seedling diseases or foot rots. Thus, the incubation period of tomato wilt (*Fusarium lycopersici*) is 35 days at a soil moisture of 37-40% of the water-holding capacity, 25 days at 43-51%, 20 days at 63-80%,

19 days at 80–90% and, when the soil is saturated, 0 days, i.e. a condition of insusceptibility is reached (Clayton, 1923). At low and very high soil moistures, the disease disposition of the plants is slight. These figures hold only for artificially created constant soil humidities; with varying soil moistures, which always occur in nature, the result varies according to the direction in which the vitality or the growth energies of the plant are modified. Plants grown in a moisture-saturated soil and resistant so long as they are rooted in this become diseased as soon as the water content of the soil is reduced, &c.

TABLE LXIX

*Influence of soil moisture on root rot of tobacco.
(After Johnson and Hartman, 1919)*

Soil water content in % of the water-holding capacity	Leaf area harvested per plant	
	Uninfected row sq. cm.	Infected row sq. cm.
25	34	9
50	81	22
75	301	89
100	239	7

The opposite case, severe disease at lower and higher levels of soil moisture but only slight disease at medium levels, is exemplified in black rot of tobacco (*Thielavia basicola*) (Table LXIX).

(b) *The Influence of Atmospheric Humidity on the Disease Proneness of the Host*

Like soil moisture, atmospheric humidity may also modify the disposition of the host to infection and to disease in certain infectious diseases of plants; some infections, for instance, tend to abort or to remain sterile in a dry atmosphere. However, since atmospheric moisture at the same time also alters the fecundity of the pathogen (p. 160) and the environmental conditions determining the success of infection (p. 27), it is often difficult to separate the different factor groups from one another.

If, for instance, a *Phytophthora* epidemic of potatoes breaks out in a rainy summer, it is probable that the high atmospheric moisture has exerted its effect in the main by favouring the pathogen. Naturally the host has been affected at the same time because excessive water-supply and a low transpiration rate have increased the susceptibility of the potato foliage. But, in this case, the decisive influence of atmospheric moisture must be attributed to its effect on the pathogen, since a *Phytophthora* epidemic may occur under conditions merely of high atmospheric humidity without any precipitation, e.g. through fog or heavy dew-formation. Thus, it suffices that the humidity requirements of the pathogen be optimally satisfied, and there is no need for simultaneous weakening of the host through excessive water-supply.

Similar difficulties obtain in the investigation of the *Monilia* disease of

apples, pears, &c. Doubtless the lenticels are opened more widely and are, therefore, more readily accessible to infection at a high atmospheric humidity than in dry air (heightened disposition to infection), but a high atmospheric humidity simultaneously improves the conditions for infection.

In spite of these restrictions there appears to be an example in which atmospheric humidity evidently increases the host's disposition to disease, viz. fire blight (*Feuerbrand*) of apple- and pear-trees (p. 49) caused by *Bacillus amylovorus*. In the experiment represented in Fig. 262 the trees were grown at a low level of soil moisture and infected through wounds with *B. amylovorus*. The trees of curve *T* were left in an atmosphere

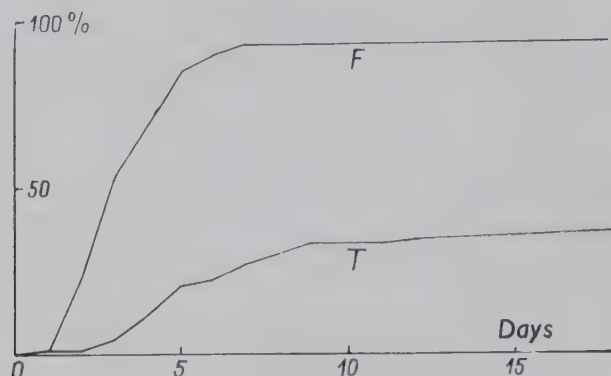


FIG. 262. The influence of atmospheric humidity on the spread of *Bacillus amylovorus* in the branches of the pear, var. Bartlett. Air temperature 24° C. Explanation in text. (After Shaw, 1935.)

of 70% relative humidity after infection, whereas those of curve *F* were transferred to a saturated atmosphere for 4 days. The abscissae in Fig. 262 indicate the number of days after infection, the ordinates the percentage length of shoots in which ordinary symptoms of wilt appeared.

In those specimens whose intercellular spaces were saturated with water vapour immediately after infection, the shoots were affected by the disease almost to the very tip (curve *F*) as against only 30% of the shoot length in the controls (curve *T*). In the latter the air in the intercellular spaces was only 99% saturated (estimated on the basis of the turgor-deficit) and the spaces were, therefore, not filled (or not permanently filled) with water of condensation. At 98% air humidity in the intercellular spaces the diseased length of shoot fell to about 5%, and below 97% there was no spread of the bacteria at all.

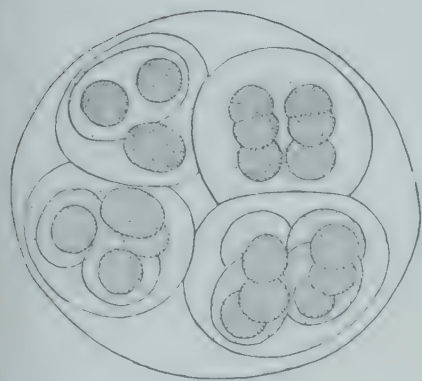
The resistance to spread in the interior of the shoot is, therefore, in this case, largely a function of the atmospheric humidity within the intercellular spaces and hence an indirect consequence of the humidity prevailing in the free atmosphere.

(c) *The Influence of its Water Economy on the Disease Proneness of the Host*

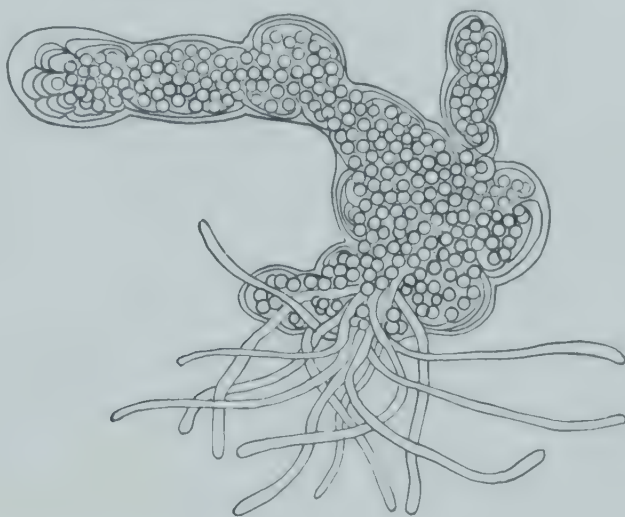
A clear correlation between water economy and disease proneness exists in the 'lichenization' of certain algae which, for different reasons (the variable factor here being the humidity of the substrate), presents similar aspects to the formation of root nodules in Leguminosae (p. 281).

On bare rocks over which water trickles there are usually patches of free algae fairly sharply separated from lichen patches. The former, especially certain species of the genera *Gloeocapsa* (Fig. 263), *Nostoc*, *Scytonema*, *Coccomyxa*, &c., frequently cover the bottom of the wet runnel ('ink stripes') as a continuous crust. They successfully resist attack from parasitic fungi, and even soredia blown or washed there are unable to develop.

Lichens are absent from these perpetually wet regions. They are confined to the drier areas wetted only by dew, rain, &c., outside the water



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FIG. 263. The influence of the humidity of the substrate on the 'lichenization' of *Gloeocapsa sanguinea*. Algal colony free from fungus from the wettest part of a water runnel. $\times 1,000$. (After Jaag, 1945.)

FIG. 264. The influence of the humidity of the substrate on the 'lichenization' of *Gloeocapsa sanguinea*. Lichenized algal colony from the dry zone outside the water runnel. $\times 250$. (After Jaag, 1945.)

courses. Here, the above-mentioned algae are attacked by certain fungi, which live at the expense of their assimilating partners whose cells become enclosed in the hyphal weft of the parasite but benefit by being protected from direct sunlight. Owing to this subsidiary effect, the parasitic attack leads to a viable consortium (Fig. 264). The parasite-host relation in these well-developed lichens resembles a chronic disease, which manifests itself in increased rate of division of the algal cells, in heightened assimilation and respiration, and also, in some ranges of forms, e.g. the *Pyrenopsidaceae*, in an hypertrophy of the host cells.

Between the area of free algae (very wet regions) and that of well-developed lichens (very dry regions) there lies a zone of moderate wetness. Here, too, the algae are attacked by fungi, which send out branch hyphae towards the green cells and drive their haustoria deeply into them (Fig. 265). This attack, however, does not lead to the formation of lichen bodies, but the algal cells affected by the parasite die and shrink into formless little lumps in their gelatinous envelopes (Fig. 266). The cells not attacked remain turgid and viable.

In the zone of intermediate wetness, the algae are thus unable to withstand the fungal attack, the association is not balanced, and a stable symbiosis cannot be established (Jaag, 1936, 1943, 1945): the fungal attack causes an acute disease, which ends in the death of the host cells.

In the lichen *Epigloea bactrospora* it is even possible to modify the character of the parasite-host relationship experimentally by altering the moisture level. In a saturated atmosphere the lichen colonies gradually

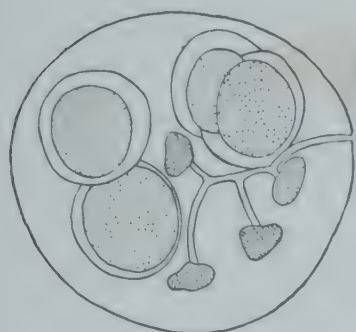


FIG. 265. The influence of the humidity of the substrate on the 'lichenization' of *Coccomyxa epigloeeae*. Algal cells with associated fungus showing deeply penetrating fungal haustoria. $\times 2,400$. (After Jaag and Thomas, 1934.)

disintegrate, as the gonidial cells rid themselves of their parasites, and continue their growth as free algae (Jaag and Thomas, 1934).

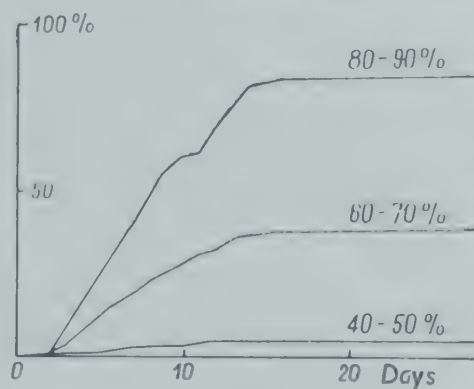
In contrast to these clear relationships found in the cryptogams, the significance of the water economy to the physiological condition in flowering plants and, therefore, to their dispositions to infection and disease is still obscure and, in any case, it is not always the same in all plant diseases (Fuchs, 1933). In addition to these factual difficulties there are others of a methodological nature, e.g. irrigation systems raise the water-supply of the host but also, at the same time, atmospheric humidity and dew formation and, with these latter, the risk of infection. On the other hand, any violent interference with the water-supply also reduces the vitality and defensive powers of the host.

In cases where, for instance, the host resistance to spread depends directly on the moisture content of its intercellular spaces, as in the fire blight of apple- and pear-trees (p. 412), an increased water-supply will certainly increase the severity of attack. In the investigation shown in Fig. 267 the experimental trees were grown at soil moistures of 80-90%, 60-70%, and 40-50% of the water-holding capacity. In the first series the average shoot-length was 30 cm., in the second 22 cm., and in the third 11 cm.; the same technique was employed as in the experiment shown in Fig. 262. With decreasing water-supply the average length of the diseased portion of the shoot fell from 84% at a high soil moisture to



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FIG. 266. The influence of the humidity of the substrate on the 'lichenization' of *Gloeocapsa sanguinea*. Algal colony from the intermediate zone partly invaded by fungus; the infected cells have been killed. $\times 1,500$. (After Jaag, 1945.)



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FIG. 267. The influence of water-supply on the spread of *Bacillus amylovorus* in the branches of the pear, var. Bartlett. Explanation in text. (After Shaw, 1935.)

4% at a low soil moisture. However, one may legitimately ask whether the drier conditions of growth had merely increased the resistance to spread, or whether they had not, at the same time, heightened the defensive power of the tissues against the bacteria and their toxins.

In some fungal diseases of trees it is even more difficult to decide how far an improved water-supply modifies the resistance of host tissues to spread, and to what extent it also affects defence readiness. The fungi in question are facultative parasites and can, therefore, live equally well in dead wood as in the bark, sap-wood, or heart-wood of living trees—especially, as we know from experience, if the water-supply of the trees has been upset. These fungi are markedly aerobic and, therefore, are immediately inhibited by a reduction in the air content of the attacked tissues below a certain limit or by an equivalent increase in water content. For instance, among the causal agents of the blue rot of conifers (Fig. 268), *Discula pinicola* grows best in the sap-wood of pines at a water content of 34%, *Ophiostoma caerulea* at 55%, *Leptographium Lundbergii* at 62%, and *Ophiostoma pini* at any level of water content from 39 to 163%; hence each of these fungi has its own particular requirements as regards water content.

The natural water content of 25-year-old pine trees averages 145% in the outer sap-wood, 152% in the inner sap-wood, and 44% in the

heart-wood. Hence, because of its high water content, the sap-wood of the healthy standing tree is protected from attack by the first three of the above-mentioned blue rot fungi. The conditions are here just the reverse of those found in pome fruit trees towards *Bacillus amylovorus*.

If such a tree is felled or its roots are damaged, this protection (at any rate against *Ophiostoma caerulea*) lasts only until the air content of the fresh wood reaches 15% of the volume. If the water content falls still farther and the volume of air rises to 42% of the volume of wood, optimal growth of the fungus results. Hence, free water is not required by the mycelium of blue rot which can thrive so long as the cell walls still contain water

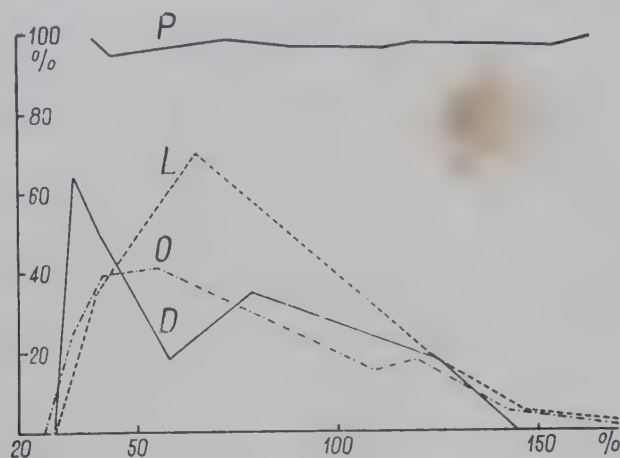


FIG. 268. Relation between the water content of pine sap-wood and its resistance to the spread of four blue rot pathogens. Curve L: *Leptographium Lundbergii*. Curve O: *Ophiostoma caerulea* (= *Ceratostomella caerulea*). Curve D: *Discula pinicola*. Curve P: *Ophiostoma pini* (= *Ceratostomella pini*). Abscissae: water content relative to the dry weight. Ordinates: discoloured timber as percentage of total volume. (After Lagerberg *et al.*, 1927.)

but, if part of this water of imbibition now disappears, fungal growth ceases owing to lack of water.

A reduction in the water-supply has a similar effect on attack by *Nectria cinnabarina* (coral spot) on the cortex of *Ulmus scabra* (= *U. montana*). In the bark of normal elm branches saturated with water in the spring, *Nectria* infection advances only 0.3–1 cm. per month. A decreased water content following injury accelerates fungal growth: with a reduction in weight of 12% the mycelium grows 4 cm. per month; with a reduction of 16%, 5 cm.; of 18%, 6 cm.; and of 32%, 10 cm. Similarly, *Nectria ditissima* is able to cause a brown discoloration of the living bark of beech over a distance of 1–3 mm. in 6 weeks when the specific gravity of the latter is 1.11–1.10, 7 mm. when the specific gravity is 1.05–0.92, 16–20 mm. if it is 0.91, and 30 mm. if it is 0.88 (Münch, 1909, 1910).

Since illustrations of water content within this range also occur under natural conditions in the course of the year, it becomes understandable why these two trees, as also *Aesculus* and other street trees, are resistant to *Nectria cinnabarina* in the period of their active vegetative growth, but highly susceptible in autumn and winter (seasonal resistance). The same

applies to poplar and *Valsa sordida*, which causes a sudden die back (apoplexy). In the same way, it is equally clear why, in forests, the over-shadowed, poorly nourished, and ill-watered trees fall victim to these fungi more readily than the better-grown trees.

The increased water content, however, doubtless protects the tissues not only directly by displacing the air necessary for the fungi, but also indirectly by increasing the vital energy of the cells. When the water content of the trunks diminishes with the natural winter dormancy or through wounds or, in street trees, through the covering of the root system by asphalt, the resistance of the cells diminishes at the same time. In summer it is not only the water content of the wood that is at a maximum but also the reaction capacity of the tissues; the increased resistance to spread and the heightened reaction proneness of the tissues mutually reinforce one another. Hence, if *Trametes radiciperda*, which causes a butt rot or 'dry red rot' of spruce, and *Stereum frustulosum*, which causes 'partridge-wood' of oak, are normally found only in the heart-wood and not in the sap-wood, it is probably not only the lower water content of the former but also its lower defence readiness that are responsible, since both fungi are able to grow for at least 10 weeks even if completely cut off from air (Bavendamm, 1928).

TABLE LXX

Influence of water supply on the susceptibility of tomato leaves, variety Lukullus, to Cladosporium fulvum. (After Volk, 1931)

Water content of soil	Mean incubation period in days	Average area of leaf spots after 20 days	Occurrence of necroses after days
%		sq. cm.	
80	12	1.8	51
50	12	1.5	58
20-25	14	1.0	71

If it is impossible to define precisely the influence of the water economy on the disposition of the host in these examples where it represents, at least in part, a direct factor in resistance to spread, how much greater are the difficulties in those infectious diseases where the water economy modifies primarily the anatomical structure and the physiological state (e.g. Tumanow, 1927), and only indirectly affects the disease disposition of the host. In these cases an abundant water supply generally raises susceptibility to eusymbiotic parasites and increases resistance to parabiotic parasites. Thus, Table LXX shows that the susceptibility of tomatoes to *Cladosporium fulvum* (leaf mould), increases with growing restriction of the water supply, whereas Table LXXI shows that the resistance of Petkuser rye to brown rust is increased by such water restriction.

6. *The Influence of Light on the Disease Proneness of the Host*

In contrast to temperature, which affects simultaneously both host and parasite, light, as a rule, affects the host almost exclusively, an exception

being the Erysiphaceae (p. 73). Light particularly influences the carbohydrate and protein metabolism and thus, on occasion, modifies the disposition of the host prior to infection and the post-infectious course of the parasitic relationship.

TABLE LXXI

Influence of water supply on the susceptibility of Petkuser rye to Puccinia dispersa (brown rust). (After Volk, 1931)

Water content of soil	Incubation period in days	Frequency of attack (% positive inoculations)	Reaction type
%			
80	9	100	4
60	10	95	4
40	11	68	2-3
20	18	42	2
Plants wilting	20	12	1

Three aspects will be considered separately: (a) intensity of light, (b) duration of light, and (c) quality of light.

(a) Intensity of light. Rust infections do not succeed if light be absent during the infection or during the succeeding 48 hours (complete darkness). The cause is probably to be found in local hyperergic necroses of host cells adjoining the focus of infection.

Lack of light tends to weaken the host and makes it more susceptible to eusymbiotic parasites and more resistant to parabiotic parasites. Papilionaceae grown in weak light lose their resistance to *Bacterium radicola* (p. 282), tomatoes grown in dark greenhouses are particularly susceptible to *Cladosporium fulvum*, and etiolated cereal seedlings to *Fusarium* diseases. Conversely, cereal plants grown in weak light are more resistant to *Helminthosporium* and rust attack: because of the tendency of the tissues to necrose the type of attack approaches the necrogenous which, in this case, is the resistant type.

As the intensity of light is increased to a higher level, both resistance to eusymbionts and susceptibility to parabionts generally increase to a definite optimum. However, different parasite-host combinations react in different ways to the same change of illumination; i.e. they show characteristic reactions to light, just as different parasite-host pairs show characteristic reactions to temperature (see p. 406). Table LXXII indicates that, at all light intensities, naked barley is 100% susceptible to *Helminthosporium* biotype 3501-11 and completely resistant to biotype H-15. Its reaction to these two biotypes is not affected by increased light intensity, although it is affected in relation to biotype 118-2. On the other hand, the response of Heines Hanna barley to these biotypes changes with increasing illumination.

A similar condition obtains in the rust fungi: the more light their host plants receive the more nearly does the form of attack approach the eusymbiotic type (reaction type 4), the greater the number of uredosori and of

spores formed in them, and the shorter the incubation period and the time to sporulation (Table LXXIII). But, as with *Helminthosporium*, this is only a rough generalization, and one can always find parasite-host pairs which do not react at all to light or react in an opposite way.

TABLE LXXII

Influence of additional illumination by means of Osram clear glass electric bulbs on the susceptibility of barley to Helminthosporium gramineum (% Incidence). Greenhouse experiment, February-March. Air temperature 10° C. (After Isenbeck, 1937)

Barley variety	Biotype	Additional illumination		
		0	40 Watt	75 Watt
Naked barley . . .	3501-11	% 100	% 100	% 100
	H-15	0	0	0
	118-2	57.6	81.2	100
Heines Hanna . . .	3501-11	30.6	40	56.7
	H-15	0	1.7	0
	118-2	5.4	33.3	44.8

TABLE LXXIII

Influence of light intensity on attack by Puccinia glumarum hordei (yellow rust) on Pannier barley. Mean air temperature 8.6° C. (After Bever, 1934)

Light intensity in candle-power	Incubation period (days)	Incidence of attack (% infected plants)
960	11	88
576	12	100
384	13	80
192	13	80
96	16	72

The influence of increasing amounts of light on the susceptibility of the host cannot, therefore, be explained shortly as a mere matter of changes in the protein and carbohydrate metabolism; much more complicated processes must be involved which affect the reactivity in the interplay of every single variety of host with each particular race of pathogen. Consequently, more detailed consideration shows that the influence of light on the different phases of the parasitic association, e.g. first to third day, fourth to sixth day, &c., may be different (Sempio, 1939).

(b) Duration of light. Under natural conditions, the length of day (photoperiod) influences the shooting of cereals and hence their smut diseases (all our cereals with the exception of maize and millet are long-day plants).

Under artificial conditions, an increase in duration of light usually reduces the susceptibility of the host to eusymbionts (Table LXXIV), and increases it to parabionts, but the various manifestations of the pathogen-

host relationship are affected differently. Thus, Table LXXV shows an optimum incubation period of 12 hours lighting, whereas the reaction type suddenly reaches the greatest resistance (abortion of the infection loci and formation of spots) with a long (15 hours) or continuous illumination (24 hours). Here, again, we are only at the beginning of our investigations.

TABLE LXXIV

Influence of duration of light on the susceptibility of tomato leaves to Cladosporium fulvum. (After Volk, 1931)

<i>Duration of light per day in hours</i>	<i>Occurrence of necroses after days</i>	<i>Diseased area after 30 days in sq. cm.</i>
3½	30	1.8
9	35	1.3
24	45	0.9

TABLE LXXV

*Influence of duration of light ('day' length) on attack by Puccinia glumarum hordei on Pannier barley. Mean air temperature 8.6° C.
(After Bever, 1934)*

<i>Length of day (hours)</i>	<i>Incubation period (days)</i>	<i>Incidence of attack (% infected plants)</i>	<i>Reaction type</i>
6	20	84	4
8	16	80	4
10	13	96	4
12	9-11	96	4
15	12	76	0
24	12	60	0

TABLE LXXVI

Influence of differently coloured light on the attack by Helminthosporium gramineum on barley. (After Isenbeck, 1937)

<i>Barley variety</i>	<i>Biotype</i>	<i>Incidence of attack when colour of light is</i>			
		<i>Red (max. in- tensity at 625 μμ)</i>	<i>Green (max. in- tensity at 525 μμ)</i>	<i>Blue (max. in- tensity at 460 μμ)</i>	<i>White</i>
Naked barley . .	3501-11	%	%	%	%
	68-1	27.2	10.2	0	52.6
	118-2	61.5	2.7	0	78.5
Heines Hanna . .		25.7	2.9	43.4	0
	3501-11	20.0	1.8	0	27.2
	68-1	0	0	0	1.4
	118-2	4.6	3.2	0	7.0

(c) Quality of light. The relation of the disposition of the host to quality of light varies with different parasite-host pairs, as it does to temperature and light intensity. Table LXXVI shows that naked barley

infected with biotype 68-1 is most heavily attacked in red or white light and not attacked in blue light, but when infected with biotype 118-2 the heaviest attack is in blue light and there is no attack in white light. Heines Hanna barley reacts somewhat differently, and is not attacked by any of the three biotypes in blue light.

7. *The Influence of the Carbon Dioxide Content of the Air on the Disease Proneness of the Host*

Within the range of carbon dioxide concentrations normal to life, higher concentrations in the air have effects similar to those of increasing amounts of light on both assimilation and the disease disposition of the host: they favour resistance to eusymbiotic pathogens and susceptibility to parabiogenic pathogens.

For instance, if there is an increase in the CO_2 content of the air from 0.03 to 0.5%, the susceptibility of tomatoes to *Cladosporium fulvum* diminishes (Volk, 1931). Carbon dioxide manuring of greenhouses, therefore, both increases the yield and reduces the disposition to disease.

On the other hand, lack of CO_2 increases the incubation period of rust diseases and infections are less successful. The normal CO_2 content of the air is sufficient to allow good infection, but the result of infection can be further improved by artificial increase of the CO_2 content. The optimal carbon dioxide concentration, as measured by the fecundity or intensity of sporulation by the parasite, lies between 0.2 and 0.7% for the most important cereal rusts (Gassner and Straib, 1929). If the carbon dioxide concentration be increased still further (up to 3-6%), infections are again less successful: no more uredosori are produced, and the host tissues react to the infection only with light-green discolorations.

At carbon dioxide levels above those normal for living organisms, both host and parasite are poisoned. But the limiting concentrations are at different levels. Growing plants are, on the whole, more sensitive to high CO_2 concentrations than are the common parasitic fungi; this circumstance is of decisive importance in snow mould of cereals (mostly caused by *Fusaria*) which, as the name implies, usually occurs where snow lies for a long time (Fig. 269). Carbon dioxide from the soil accumulates below the encrusted snow blanket and affects the host and pathogen differently (Lundegårdh, 1923).

The host plants are damaged by the excessive carbon dioxide concentration. Wheat germinates best at the normal CO_2 content of the air, i.e. 0.03%. Even at 1-1.5% germinability begins to fall, and above 2% the lengths of coleoptile and root are noticeably reduced. Contents of over 1% do actually occur under outdoor conditions below a slowly melting snow cover, and even without snow at a normal depth of sowing if there is poor aeration and a high water content of the soil.

The effect of CO_2 on the fungi now to be considered is different (Braun, 1932; Klaus, 1943). The growth of *Gibberella Saubinetii* and *Fusarium culmorum* is actually stimulated by carbon dioxide contents of 2-7%, while

F. avenaceum and *F. herbarum* do not react to contents below 5%. Hence, on the one hand, the vitality of the host plants is diminished by super-normal carbon dioxide concentrations and, on the other hand, the growth-rate and probably also the aggressiveness of at least two of the more important pathogens are increased. Consequently, wheat seedlings germinated in soil containing *Gibberella* and kept during the first 9–12 days in an atmosphere containing 2–8% carbon dioxide concentration, show a snow mould incidence of 100% as against 0% in normal air.

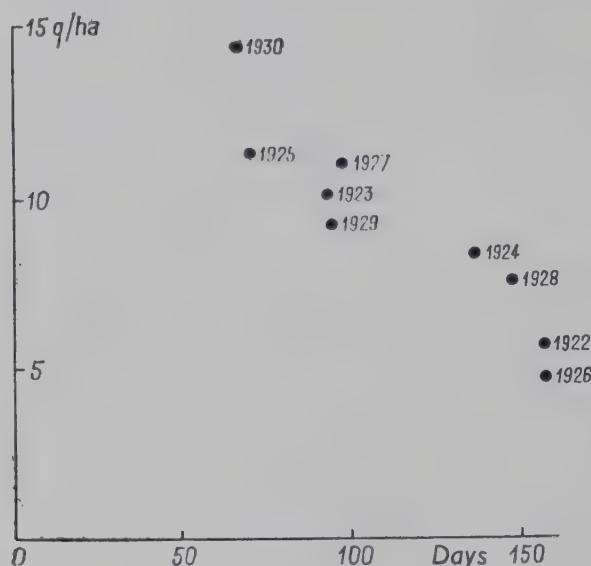


FIG. 269. Correlation between the persistence of the snow cover and the damage caused by the snow mould to winter rye in Latvia. Abscissae: duration of the snow cover in days. Ordinates: mean yield in q./ha. (After Eglits, 1931.)

On the side of the pathogen, the conditions necessary for a severe attack of the disease coincide with those required by *Herpotrichia nigra* (Fig. 159). *Gibberella* does not 'prefer' the low temperatures but can make use of them if the spring sunshine warms the slowly melting and caking snow cover, all the more because it is growing at optimal humidity (as soon as the snow melts, the external mycelia quickly collapse but, when first exposed, they are visible, resembling a spider's web).

On the side of the host, however, the conditions differ from those in *Herpotrichia* attack. Under the snow the latter also occurs on normally vigorous plants, whereas snow mould is confined to enfeebled individuals. In contrast to *Herpotrichia*, *Gibberella* is able to grow only with difficulty at low temperatures (Fig. 254) and would hardly be able, in this state, to overcome normally vigorous plants. Hence, its mode of life under the snow borders on debility parasitism.

In contrast, however, to *Herpotrichia*, snow mould is not the only disease of cereals caused by Fusaria, and others can occur without any snow covering, e.g. foot rot of young plants or *Fusarium* blight of the ears (Fig. 229). In these cases the host need not be enfeebled but, if the attack is to be sustained, the conditions for the pathogen must be nearly

optimal, i.e. the soil must be heavily contaminated (Fig. 38) and soil humidity and soil temperature (Fig. 253) must attain optimal values.

8. *The Influence of Soil Characteristics on the Disease Proneness of the Host*

The soil with its physical, colloid chemical, and chemical properties, &c., defines the conditions of existence both for the pathogens living in it (suitability as germ reservoir for the maintenance of parasites; p. 95) and for the host plants rooted in it. Its importance for the vigorous growth and, hence, indirectly for the physiological disease resistance of the host plants, is widely recognized in crop husbandry and has been fully described, for instance, by Braun (1937).

TABLE LXXVII

*Correlation between soil quality and attack of spruce by *Trametes radiciperda* (annosus butt rot). (After Falk, 1930)*

Disease incidence	Site class						
	I/II	II	II/III	III	III/IV	IV	IV/V
	%	%	%	%	%	%	%
Number of trees infected with butt rot:							
1-30% . . .	20	45	57	75	88	100	100
More than 30% . . .	80	55	43	25	12	0	0
Proportion of rotten wood:							
1-10% . . .	67	23	45	68	64	67	
More than 10% . . .	33	77	55	32	36	33	

At times, however, opinions differ greatly with regard to particular problems. It is not even possible to give a generally valid definition of a 'good' soil; for instance, the susceptibility of the spruce to butt rot increases with the 'quality' of its site. In Table LXXVII several hundred spruce plantations have been grouped into five 'site' classes on the basis of rate of increase (mass yield), *I* being the best and *I'* the worst. In the first line, the percentage of plantations with 1-30% of attacked trees (i.e. low disease incidence) is given and, in the second line, the number of plantations showing high incidence of disease. It is clear that the incidence of attack rises with the 'quality' of the site. However, the quantity of rotten wood does not vary with the goodness of the site, which means that infection does not advance so far up the trunks in the good site classes. Therefore, soil quality affects the incidence of attack inversely to the intensity of the disease course: the better the soil the greater is the number of diseased trees, but also the more restricted the extent of disease within the individual trunks.

This example demonstrates once more the complexities of the problem of disposition: conclusions will differ according to the criteria adopted.

Moreover, the forester, like the farmer, must judge the value of locality from the standpoint of human utility (amount of increase of production), but it may well happen that the more slowly growing plants are, in reality, stronger than specimens growing more quickly.

TABLE LXXVIII

Influence of physical properties of the soil on the occurrence of tuber rot of potatoes. (After Jones et al., 1912)

Thickness of soil layer (cm.)	Number of diseased tubers	
	Clay soil	Sandy soil
	%	%
1.25	100	57
7.5	96	48
15.0	61	30

The effect of each particular soil characteristic on the disease disposition of the host plants naturally varies according to the pathogen-host pair.

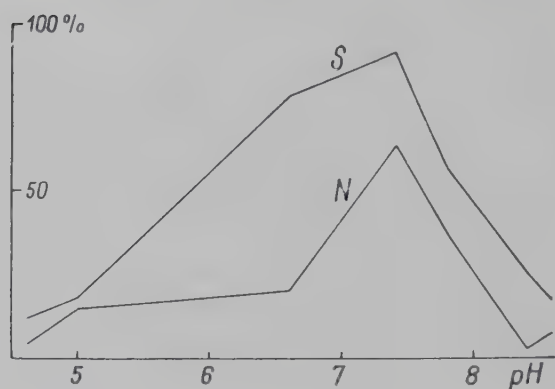


FIG. 270. The influence of the soil reaction on the attack of two species of oats by *Ustilago laevis* (covered smut). Curve S: *Avena sativa* (var. Victor). Curve N: *A. nuda* (var. *inermis*). (After Reed and Faris, 1924.)

Jones *et al.* (1912) examined the influence of the physical structure of the soil on the susceptibility of potatoes to *Phytophthora* by burying healthy tubers at different depths in clay or sandy soils; on top of the soil they then placed diseased foliage, which was heavily watered for several days. Tubers are infected by conidia washed down from the leaves, hence one would expect that under the conditions of the experiment more infections would occur in the sandy soil with its greater porosity than

in the more compact clay. Table LXXVIII shows an opposite result; the clay soil either provided better conditions for infection because of its physical properties, or it heightened the disposition of the tubers to *Phytophthora* either by reducing cork formation (Löhnis, 1925) or by lowering their respiration rate.

The soil reaction may, in some cases, alter the disposition of the host by influencing its nutrition and the degree of swelling of its membranes but, as the pathogen is affected at the same time and in the same way, it is often scarcely possible to apportion the effect between the partners. On page 34 we attempted to explain the reduction in attack by club root with increasing alkalinity of the soil solution mainly by inhibition of the pathogen but, in the disease shown in Fig. 270, it is necessary to take into consideration a variable disposition of the host itself as well as differential effects on the germination and development of the pathogen.

Improved soil aeration favours attack of cereals by foot rots and eyespot but inhibits *Fusarium* diseases. An increasing humus content of the soil over a range from 0 to 5% appears to favour black leg of sugar beet seedlings; but it appears to reduce the disposition of spruce to butt rot up to a certain optimum. In the absence of other possible interpretations, even the 'earth rays' (i.e. physically not yet defined, stimulatory zones, to which diviners respond physiologically) have been held responsible for the local disease dispositions of certain plant stocks, but subsequent examination has not confirmed the suggested connexion (e.g. Gäumann, 1935, 1936; Rohmeder, 1937).

9. *The Influence of Grafting on the Disease Proneness of the Host*

Where there is disharmony between stock and scion the metabolism and vitality of the latter suffer. Thus, the so-called 'degeneration of varieties' in fruit trees is caused (apart from the introduction of new races of pathogens) mainly by grafting the particular varieties on to non-congenial stocks.

The stock may affect the disease disposition of the scion in the following ways:

1. It may directly alter the metabolism of the scion. If, for example, certain varieties of pear are grafted on to unsuitable quince stocks, they tend to become chlorotic and thereby immune to *Gymnosporangium Sabinae* (cluster cup rust). Conversely, the susceptibility of tomato and potato foliage to *Phytophthora infestans* (late blight), and of tomato leaves to *Cladosporium fulvum* undergoes no measurable change when different Solanaceae are used as stocks (Bond, 1936). Tomato on *Datura Stramonium* stock is an exception because the incubation period of *Cladosporium fulvum* rises from 9 to 11 days and severe dark-coloured necroses appear instead of the usual symptoms (Volk, 1931).

2. The stock can influence the disease disposition of the scion directly by evoking in it an induced tolerance of certain viroses (p. 342).

3. The stock can influence the disease disposition of the scion indirectly by altering the vigour of its growth and the maturation of its tissues. The wood of pome and stone fruit trees matures less completely on strongly growing stocks, hence its frost-hardiness diminishes (Loewel and Schubert, 1941; Hilkenbäumer, 1942) and the susceptibility of the trees to wound parasites becomes greater (e.g. the susceptibility of apple trees to *Nectria galligena*).

4. In periclinal chimaeras a resistant skin may protect a susceptible core so far as simple resistance to penetration is concerned. Even where infection takes place through the stomata, as in the *Septoria* disease of tomatoes, experimental results are in good agreement with the assumption of a periclinal chimaeral nature of the graft hybrids; and it is apparent that, in spite of their intimate association, one partner does not affect the susceptibility of the other (Table LXXIX). Sectorial chimaeras of the corresponding types behave similarly (Jørgensen, 1928). But, on the other

hand, where the character of the graft hybrids is not clear, as in burdo formation (nuclear fusion of the two partners at the site of grafting), or in true periclinal chimaeras as in the *Crataegomespili* and *Pirocydoniae*, the conditions of disposition to rusts (*Gymnosporangium* spp.) and to powdery mildew (*Podosphaera oxyacanthae*) are obscure (Sahli, 1916; Maurizio, 1927).

TABLE LXXIX

Susceptibility of some periclinal chimaeras of Solanum spp. to Septoria lycopersici. (After Klebahn, 1918)

<i>Variety</i>	<i>Skin</i>	<i>Core</i>	<i>Course of disease</i>
<i>S. Lycopersicum</i> (tomato)	Tomato	Tomato	Susceptible
<i>S. nigrum</i> (black nightshade)	Black nightshade	Black nightshade	Resistant
<i>S. Koelreuterianum</i>	Epidermis of black nightshade	Tomato	Severe attack with formation of pycnidia; the resistant epidermis has not protected the susceptible core
<i>S. Gaertnerianum</i>	Two layers of black nightshade which may multiply irregularly by subsequent divisions	Tomato	Variable; at least two layers are resistant. The surface layers can, therefore, sometimes prevent the parasites from penetrating
<i>S. tübingense</i>	Epidermis of tomato	Black nightshade	Slight formation of spots without pycnidia
<i>S. proteus</i>	Two layers of tomato	Black nightshade	Infection and formation of pycnidia nearly as severe as in pure tomatoes

10. *The Influence of Primary Diseases on the Disease Proneness of the Host*

In some cases there is no manifest relation between initial infection of a host by a given pathogen and super-infection by other pathogens, e.g. yellow rust (*Puccinia glumarum*) and powdery mildew (*Erysiphe graminis*) may occur simultaneously on cereals without visibly influencing one another. Other pathogens, however, modify the reaction norm of the host to a super-infection; premunity (p. 307) and infection-bound tolerance (p. 343) are examples in which a given pre-disease modifies the reaction norm of the host to super-infection by the same or a related pathogen. The present sub-section deals with the parasitogenic changes in susceptibility to unrelated pathogens.

In certain infectious diseases of plants these changes occur so regularly that, in the course of a given disease, the same sequence of primary pathogens and secondary parasites almost always recurs. But as the latter frequently dominate the clinical picture and bring about the disappearance

of the primary pathogen, a superficial examination reveals only the secondary co-parasites which are thus liable to be regarded as the actual pathogens.

A disease which until very recently was described incorrectly in textbooks and handbooks may serve as an example. In the Mediterranean region a Phycomycete, *Phytophthora cambivora*, causes ink disease of chestnut, &c. The fungus grows from the soil into the roots and from there into the cambium under the bark of the trunk, and weakens the tree by destruction of the phloem, or kills it (Petri, 1917). This primary infection is almost always followed by secondary parasitic fungi which grow more or less deeply into the wood and up the trunk, attack the branches, and help materially in the rapid destruction of the tree. Among them are *Melanconis perniciosa* (= *Coryneum perniciosum*), *Sporotrichum laxum*, and *Torula nivea*. These three secondary parasites are so characteristic of the clinical picture that each of them in turn has been regarded as the cause of ink disease and, even to-day, is still treated as such in some accounts.

According to the point at which the parasitogenic change of disposition occurs in infectious diseases of plants, changes of infection disposition and changes of disease disposition may be distinguished.

The infection disposition to eusymbiotic parasites is probably always increased by a pre-disease. Thus the primary parasite may break down the barriers of resistance to penetration and thereby open the way into the interior of the host for the secondary parasites (p. 56). *Endostigme inaequalis*, when it occurs as twig scab of the apple tree, forms an entry point for the pathogen of apple canker (*Nectria galligena*), and when it occurs as fruit scab forms an entry point for the *Fusaria* and *Penicillia* which cause rotting. When *Melampsorella caryophyllacearum* causes stem canker in white fir, the cracks gradually produced in the bark afford entrance to wood parasites such as *Polyporus Hartigii* (p. 57), &c.

In some other infectious diseases of plants, the primary pathogen not only breaks down the host's resistance to penetration but also its resistance to spread, thus making possible for the secondary parasites not only entry but also affording them, by a local change of the substrate, a start for their further extension. Thus, *Fusarium caeruleum* cannot seriously attack healthy tissue of potato tubers under normal environmental conditions (even where the skin has been injured), but a local blight lesion (Fig. 271) caused by *Phytophthora infestans* may provide a portal of entry and create a local disposition to semi-saprophytic micro-organisms like this *Fusarium*. Its growth on the blighted tissues gives it the necessary vitality to attack healthy tissues, and to cause a dry rot during winter storage.

These local possibilities of obtaining a start as a result of pre-disease of the host tissues are important in the rotting of timber. Initial rots such as may be caused by blue rot fungi (species of *Ophiostoma*, &c.; p. 416) or by *Coniophora cerebella* (cellar fungus) can prepare the substrate for the germination of the basidiospores of secondary fungi which cause destructive rots, e.g. *Merulius lacrymans* (dry rot); the initial rots thus prepare

the way for the destructive rots to colonize the healthy wood which they cannot attack unaided.

Parasitogenic changes in infection disposition are mainly a problem of plants since resistance to penetration and to spread are chiefly of importance in botany. On the other hand, the parasitogenic changes in disease disposition were first studied in human and veterinary medicine. The relation between the primary pathogen and super-infection may be non-specific or specific.

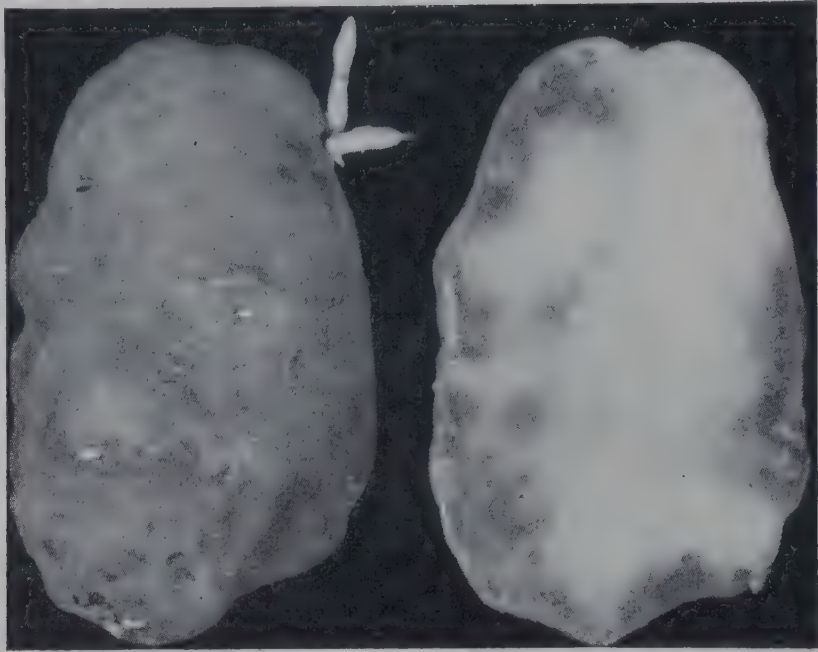


FIG. 271. *Phytophthora* diseased potato tubers. Left: external appearance, areas affected by the dry rot have collapsed; the tuber has formed emergency sprouts. Right: longitudinal section showing the brown dry rot loci. Approx. $\times \frac{3}{4}$. (After Maag, 1944.)

The least specific case is that in which the primary disease increases the disposition of the organism to any subsequent diseases, as measles increases the human disposition to pneumonia, tuberculosis, &c.

Specific parasitogenic disease disposition is found in swine fever. The primary disease is a virosis, but the course of the illness is determined by the super-infection of a co-parasite, *Bacillus suipestifer*. This bacillus may, on occasion, have been present in the body prior to the virosis, but it could only become pathogenic after the virosis had created the corresponding disease disposition. *Bacillus suipestifer* is, therefore, characteristic of the final stages of swine fever as *microbe de sortie*. Hence, as soon as its presence is established there is strong support for the suspicion that it was preceded by the specific virus. The probable existence of a definite but unsuspected primary disease is, therefore, indicated in this case by the specific and manifest secondary illness.

The same possibilities of non-specific and specific changes of disease disposition occur among infectious diseases of plants. As in medicine, so also in plant pathology the non-specific changes of disease disposition

are much the more frequent. In contrast to the raising of defence potential by specific sensitization (p. 306) a heightening of susceptibility is the most common trend of non-specific change in disease disposition. Wheat plants containing bunt mycelium (*Tilletia tritici*) show a heightened susceptibility to yellow rust (*Puccinia glumarum*) after they have started to ear and especially after flowering. They do not show this heightened susceptibility in the seedling or juvenile stages, nor do they show it unless they belong to a variety of a labile and intermediate reaction type (Table LXXX). Highly susceptible varieties remain highly susceptible (reaction type 4) and resistant varieties usually remain resistant (reaction type 0).

TABLE LXXX

Influence of bunt infection on the behaviour of wheat to yellow rust between earing and flowering. (After Straib, 1938)

Wheat variety	Biotype 2		Biotype 7	
	Not infected with bunt	Infected with bunt	Not infected with bunt	Infected with bunt
Michigan Amber . . .	4	4	4	4
Strubes Dickkopf . . .	0	3-4	2	4
Carstens V	0	0	3	3

Eyespot and foot rot diseases of cereals (Figs. 92 and 91) are caused primarily by *Cercospora herpotrichoides* and *Ophiobolus graminis*, but the extent of the damage is often determined by the secondary parasites associated with them. These are *Wojnowicia graminis*, *Leptosphaeria culmifraga*, *L. herpotrichoides*, and several *Fusaria*, which may sometimes produce wholly atypical clinical pictures, depending on their combination and the time of their admission. In extreme cases, foot rot may lead to a parasitogenic susceptibility to debility parasites. Foot rotted cereals are often attacked by black moulding fungi, *Gladosporium herbarum*, &c., which never establish themselves on normally vigorous individuals.

More rarely the change of non-specific disease disposition tends towards a reduction of susceptibility. Such an antagonism exists, under certain circumstances, between bunt and powdery mildew: plants of the wheat variety Mentana containing bunt mycelium become resistant to powdery mildew (*Erysiphe graminis tritici*) (Sempio, 1938). Their disease disposition has undergone a change directly opposite to that for yellow rust.

An example of specific change of disease disposition among plants is shown by the twist disease of wheat (Fig. 272) (*Dilophospora alopecuri*, = *D. graminis*). The German name 'Federbuschsporenkrankheit' is due to the tufts of bristles at both ends of the pycnospores. The fungus cannot colonize either healthy (intact) or injured wheat plants (Atanasoff, 1925) but it can colonize intact rye seedlings (Schaffnit and Wieben, 1928). However, wheat can become susceptible following an attack of eelworm disease. The eelworms not only make the plant susceptible to the fungus, but they transmit it (Fig. 273), and convey it to the growing-

point of the host. This penetration to the growing-point which is necessary for successful infection cannot occur without nematodes. Conversely, the susceptibility of wheat plants to the fungus persists only so long as they are inhabited by nematodes; as soon as these migrate, die, or encapsulate themselves, the further spread of the fungus ceases. The control of the nematodes thus means the control of twist disease. Since twist is a typical secondary disease, its appearance in wheat entitles one to conclude (as in



FIG. 272. Twist disease of wheat. Left: partial degeneration of the ear with formation of a sclerotium-like crust. Centre and right: deformations of the ear owing to its tip being arrested in the leaf sheath. Original $\times \frac{3}{4}$.

FIG. 273. Larva of *Tylenchus tritici* to which spores of *Dilophospora alopecuri* have attached themselves. (Diagrammatic after Atanasoff, 1925.)

the instance of swine fever) that the existence of the corresponding primary disease, in this case the nematode contamination, is highly probable.

However, in twist disease the relations between the secondary and the primary pathogen are less harmonious than in swine fever. While the nematodes, like the swine fever virus, enable *Dilophospora* to attack the host, the co-parasite now destroys whatever the nematodes have not yet destroyed and thereby becomes harmful to its pacemakers, the nematodes themselves, hindering their further spread and gall formation. For its colonization of the host, the co-parasite depends on the primary pathogen which it subsequently exterminates.

11. *The Influence of Lesions on the Disease Proneness of the Host*

In human medicine dust diseases increase the disposition of miners to tuberculosis of the lungs, and occupational eczema increases the disposition

of tar and paraffin workers to specific types of cancer. Botany also provides a series of examples in which acute or chronic lesions modify the proneness of the host to certain infectious diseases. A few possible correlations of this kind have been mentioned previously, for instance, injury as a condition of wound infection (p. 54) and depression of vitality as a cause of heightened disease proneness (p. 379). The present sub-section adds a few more groups of causes: sunburn, frost, electrical discharges, and narcotics.



FIG. 274. Beech tree injured by sunburn, subsequently infected by *Polyporus squamosus*. $\times \frac{1}{5}$. (Original photograph C. A. Burckhardt-Zwicky.)

Damage from sunburn is mostly found on the south and west sides of forest trees which have been abruptly exposed by the sudden removal of wood cover, and cannot withstand the effect of the local heat. Semi-saprophytic, wood-destroying fungi, such as *Lenzites sepiaria* which causes a red rot of conifers and *Polyporus squamosus* the agent of a white rot of deciduous trees (Fig. 274), then enter the cracks in the bark, and obtain a foothold in the damaged tissues, whence they later invade the sound mature wood. Such infections border directly on wound parasitism.

Damage by frost, chiefly due to early frosts in the case of incompletely matured shoots and to late frosts in that of premature sprouts, enhances the disposition of street trees to coral spot disease (*Nectria cinnabarina*), that of beech trees to canker (*N. ditissima*), and that of spruces to die-back (*N. cucurbitula*). In certain localities these frost-aided infectious diseases are so rife that the forester talks of special frost patches.

Cold winters occasionally increase the disposition of forest trees to other *Nectria* spp. which normally are not pathogenic to any appreciable extent, and these may sometimes cause surprising, although temporary, local

epidemics. For example, in the summer of 1940, forest beeches in eastern Switzerland died at an alarming rate, and the disease spread further in subsequent years (Leibundgut and Frick, 1943). In all the trunks examined the annual ring for 1939 was quite normal but, beginning with 1940, there was damage to the cambium, and breaking of the annual rings into wedges, followed by infection with *N. coccinea*. The injuries were a consequence of the severe winter of 1939–40 when temperatures fell to -35°C .

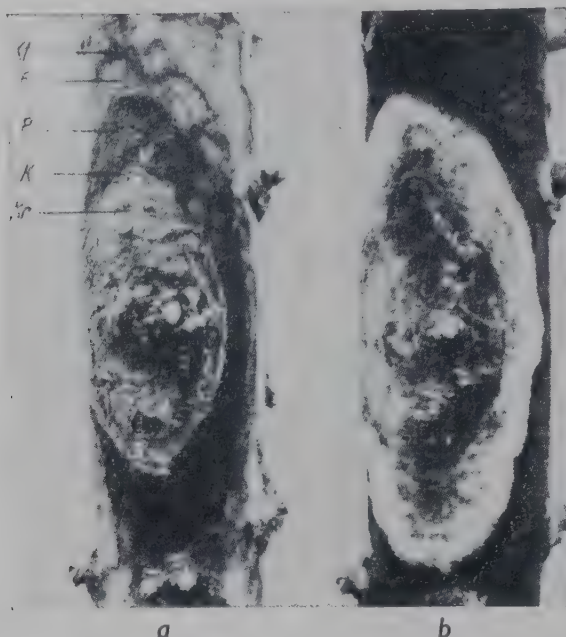


FIG. 275. Canker of the larch caused by *Dasyscypha Willkommii*. (a) Immediately after removal of the epidermis. *Kf* zone of conflict between the toxins of the pathogen and the necrogenous substances derived from the injured host cells on the one hand and the intact bark tissues on the other. *E* zone in which the bark tissues have become hyaline. *P* zone of active living mycelium of the parasite. *K* corky demarcation zone of the previous season. *Kr* bark tissue invaded by fungus the previous year. (b) The same canker after several days storage in a moist atmosphere. The living fungus mycelium in zone *P* has grown out through the surface.

Approx. $\times \frac{3}{4}$. (After Langner, 1936.)

The fungus caused the affected trees to dry up completely within a few weeks of their becoming green in the spring, although normally it does not play any noticeable role as a disease agent in the region concerned. Hence, the local frost injuries in the severe winter of 1939–40 had decisively increased the disposition of the beeches to this fungus.

The contributory effect of injury due to low temperatures is even more general in larch canker (*Dasyscypha Willkommii*) than in this case of beech trees. Cracks due to frost, wounds, animal attack, &c., may serve as initial portals of entry. The fungus is common in the Alps as a saprophyte on the shed branches but, for climatic reasons, is mostly harmless to living trees. As long as the trees are in sap they can ward off the fungus and, outside the growing season, air temperatures also are too low for it so that it cannot develop.

In the foothills, however, during spells of mild weather in the dormant

period, i.e. in late autumn and early spring, the fungus penetrates, partly parasitically and partly saprophytically, through the bark to the cambium. Here its metabolic products diffuse several millimetres deep into the healthy tissues and, reinforced by necrogenous substances, stimulate the cambium to form concentric occluding ridges. Furthermore, they damage the intact chlorophyll-containing tissues of the bark (which results, for instance, in a peculiar glassy discoloration) but do not usually kill them, although they interrupt their vegetative rhythm so that they no longer become dormant in autumn. The strips of tissue affected in this way freeze in the course of the winter, and thus there appears a severely

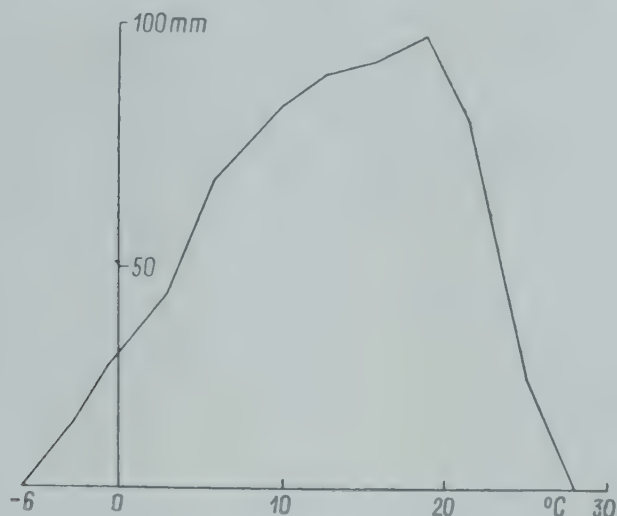


FIG. 276. The influence of temperature on the growth rate of *Crumenula abietina* (epidemic die back in alpine plantations). Ordinates: mean diameter of agar cultures after 9 weeks. (After Ettlinger, 1945.)

injured but fungus-free zone *E* (Fig. 275). In the very early spring, with the return of milder weather, the fungus enters this area more or less saprophytically, usually by way of the cambium; thus each spring it begins the disease cycle in a new zone (Langner, 1936).

This type of canker with its rhythmic sequence of necrotic depressions enclosed in concentric occluding ridges arises, therefore, through the constant reciprocal action of fungus and frost. The fungus stimulates local meristematic activity of the host, kills the weakened cells, induces wound reactions, and inhibits the neighbouring bark tissue from becoming fully frost resistant. In some cases the frost creates the initial portal of entry for the pathogen and later finishes off those host tissues which have been weakened and thrown out of their vegetative rhythm by the pathogen.

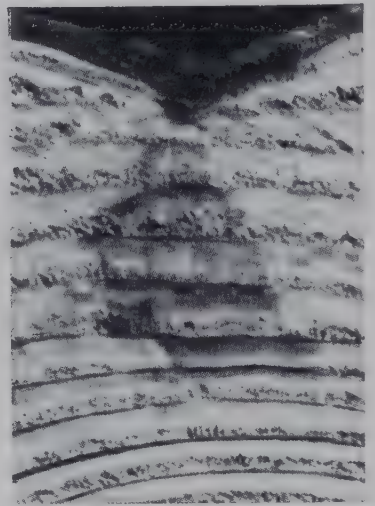
The epidemiological conditions for larch canker, compared with those for die back in alpine plantations and for *Herpotrichia*, are as given below. All three produce chronic infections, whose typical courses run for at least some years.

The pathogen of larch canker can spread in the host only during the dormant period; in the growing season it is checked by the defence reactions of the host tissues. In the Alps, however, the winter air temperatures

above the snow cover are too low for it, hence, in this situation it is generally absent as a parasite. It is, however, a ubiquitous saprophyte thriving in summer and winter (under the snow) on dead branches. In the milder foothills it can develop epidemically, as here, outside the growing period, air temperatures above 3–4° C. prevail for weeks and even months at a time. These temperatures are not high enough to break the winter dormancy of the trees, whose cellular defence reactions, therefore, remain latent; but they are high enough to permit the pathogen to grow and spread.



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FIG. 277. *Pleurotus* disease of firs damaged by lightning. Advanced stage with deep atrophied grooves. Approx. $\times \frac{1}{10}$. (After Gäumann and Jaag, 1937.)

FIG. 278. *Pleurotus* disease of firs damaged by lightning. Section through a lesion about 2–3 years old. Somewhat reduced. (After Gäumann and Jaag, 1937.)

The conditions for epidemic die back of *Pinus* spp. in alpine plantations are almost identical (p. 142). Its pathogen, *Crumenula abietina*, can also spread to any considerable extent only in the non-reacting branches and trunks of the host during the dormant season. Hence, the crowns of trees above the alpine snow cover are inaccessible to it but, as it is even more cold resistant (Fig. 276) than *Herpotrichia* (Fig. 159), it develops in the Alps during the long winter months under and in the snow in low-lying branches or in young trees (plantations). Moreover, in the foothills, during the milder months of the dormant season, it attacks the crowns of standing trees for the same reasons as does larch canker.

Finally, in the case of *Herpotrichia nigra* (p. 232), the continuous high air humidity, not the environmental temperature, is the decisive limiting factor. Hence, in contrast to epidemic die back, the attack on trees under the snow is not conditioned by a seasonal depression of their reactivity, but purely physically by the high air humidity under the snow cover, which the pathogen can make use of because it is tolerant of cold.

Like frost injury, damage caused by electrical discharges often cannot be identified as such directly, but only indirectly because of the occurrence of an unusually high local frequency of infectious diseases. This applies

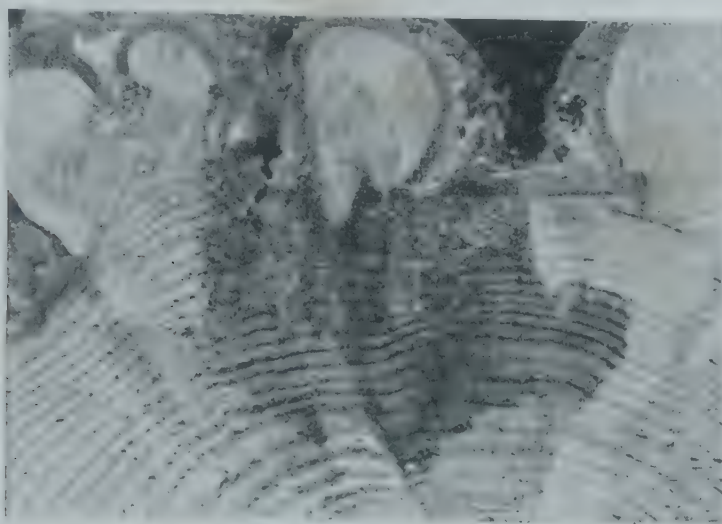


FIG. 279. *Pleurotus* disease of firs damaged by lightning. Late stage, more than 15 years old. The cambium has died in several strips but continues to grow between these strips with great tenacity, thus giving the clinical picture shown in Fig. 277. The red rot has penetrated deeply into the main wood. Approx. $\times \frac{1}{4}$. (After Gäumann and Jaag, 1937.)



FIG. 280. The influence of pre-infectional ether narcosis on the disease disposition of sugar beet leaves to *Phoma betae*. Left: 0.2 c.c. ether in 1 litre air; treatment 24 hrs. Centre: 0.01 c.c. ether in 1 litre air; treatment 8 days. Right: control (infection without narcosis). Approx. $\times \frac{3}{4}$. (After Volk, 1931.)

especially to such discharges as 'ball lightning' which in woods, for instance, affects indiscriminately all the trees over areas 50 to 100 m. diameter, and renders them accessible to unusual infections. But, from the nature of the case, it is not possible to obtain conclusive experimental proof in this group of pathological dispositions. Hence, electrical discharges can be

regarded as a cause of the heightened disposition with only a greater or lesser degree of probability.

Certain stands of silver fir (*Abies pectinata*) and of spruce (*Picea excelsa*) growing in central Switzerland along the routes of frequent hail-storms and which, because of local conditions, are doubtless exposed to strong electrical discharges, often show along their trunks a noticeable depression of the bark, followed by infection with *Pleurotus mitis* (Fig. 277). Apparently strips of cambium were first damaged or killed by the electrical discharges and thus became accessible to *Pleurotus* infection. Admittedly *P. mitis* can also attack uninjured trees, but the increased frequency of disease in stands exposed to storms, and its characteristic course, can be explained only by assuming primary injury by electrical discharges.

TABLE LXXXI

Influence of ether narcosis for 12 hours on the attack of beans by Uromyces appendiculatus (bean rust). (After Minkevicius, 1932)

Treatment	Narcosis before infection		Narcosis after infection	
	Increase of leaf area in %	Number of lesions per sq. cm.	Increase of leaf area in %	Number of lesions per sq. cm.
1. No narcosis, not infected	835±91.0	0	429±32.0	0
2. Narcotized with 386 c.c. ether, ¹ not infected	789±56.3	0	416±43.6	0
3. No narcosis, infected	700±77.6	5.6±0.59	426±40.9	3.7±1.04
4. Narcotized with 193 c.c. ether, ¹ infected	597±33.9	6.3±0.62	351±29.0	3.4±0.98
5. Narcotized with 386 c.c. ether, ¹ infected	615±53.3	6.1±0.51	309±19.0	2.4±0.69
6. Narcotized with 580 c.c. ether, ¹ infected	535±43.5	3.8±0.34	267±27.3	1.5±0.65

¹ Per volume of air of 800 c.dm.

In contrast to injuries by sunburn, frost, and electrical discharges, the problem of the influence of narcosis on disease disposition of the host is of purely theoretical interest. In the human body no measurable increase of disposition to infection or to disease seems to occur during or after narcosis. In some plants, however, metabolism is profoundly influenced by deep narcosis. Physiologically this is expressed, for example, in an increase of their nitrogen or protein content and, parasitologically, in a change of their disease proneness which is always in the direction of a heightening of susceptibility. However, this increase of susceptibility may express itself in opposing ways according to whether the pathogen-host pair be eusymbiotic or parabiotic.

In the case of eusymbiotic pathogen-host pairs, a heightening of disease susceptibility shows itself directly in an increase both of incidence of attack and severity of disease. For instance, the susceptibility of cauli-

flower to *Alternaria brassicae* (dark leaf spot) is generally only slightly affected by ether, alcohol, or chloroform narcosis before or after infection. But, when a change occurs, this is in the direction of increased susceptibility, e.g. a 48-hours concentrated chloroform narcosis after infection increases the average number of lesions per sq. cm. from one to five (Minkevicius, 1932). In other instances severity of the disease is also increased (Fig. 280).

Where pathogen-host pairs tend to exhibit a parabiogenic character, as in the rust diseases, the increase in susceptibility caused by narcosis may first show itself in a corresponding change of reaction type. For example, in Rümkers summer Dickkopf wheat infected with *Puccinia tritici* biotype 14, under the influence of a pre-infectional chloroform narcosis, the reaction type goes up from mainly 3 to mainly 4, and in Mediterranean wheat, from mainly 0 to mainly 2-3 (Gassner and Hassebrauk, 1938). In other cases the increase in susceptibility due to narcosis leads to hypersensitivity and thus to hyperergic repulse of the parasite. Table LXXXI shows that the incidence of attack on beans by bean rust falls with intensification of narcosis, regardless of whether the treatment occurred before or after infection. The damage to the host, which is manifested in the inhibition of growth of the leaves, apparently induces an enhanced readiness to necrogenous reaction and thereby a reduction of the disposition to infection.

CHAPTER 5

THE DISEASE

THE preceding chapters have been concerned with the establishment of infection and the action-potential of the pathogen; we now turn to the counter-manifestations of the host.

The bodily functions of an organism do not constitute its life, they are merely the conditions and consequences of its life to which they are subsidiary. In their totality they form the inner conditions of the life of the organism. An organism is healthy so long as the inner conditions of its life can adjust themselves harmoniously to environmental influences. If this inner equilibrium be disturbed, illness of the body ensues.

Thus every disease originates primarily in a functional disturbance of the organism. On the basis of its capacity for adjustment the organism 'seeks' first to retain and to continue its normal development and metabolism; if a disturbance occurs, it 'endeavours' to return to its norm and to reinstate its threatened equilibrium. This return to the norm is termed healing or recovery.

Accordingly, only those individuals become diseased which react to disturbing influences, i.e. deviate from their normal functional course; the bite of a cockchafer, for example, evokes no such reactions and, therefore, does not cause a disease but merely an injury to the organism.

Among the disturbances induced by external factors, infectious diseases occupy a special place, because in them not only the host but also the disease-evoking (pathogenic) agency is a living organism with a plastic range of variation in its vital requirements, actions and reactions. Disease is, therefore, a problem of mutual conflict.

In this conflict how far does the pathogen 'impose its will' on the host and how far the host its 'will' on the pathogen?

According to the general conception it is the pathogen that acts or operates, and the host that responds, 'disputes' with the pathogen or reacts; the infectious disease is considered to be the reaction of the host organism to infection! With every action, however, the pathogen changes its own substrate, thereby creating a new situation which compels it to further action; the host, by reacting, becomes biologically different to what it was before since its life is now pursued under the altered situation of infection. The response of the host, therefore, simultaneously implies action on the pathogen; the latter must adapt itself to the new reactive situation, that is to say, it must itself respond with reactions which will again call forth new reaction-actions from the host, and so on, in such a way that the pendulum of causality swings to and fro in unbroken sequence. Thus, in the course of the disease, it is not only the host that is continually

becoming different from what it was formerly, and especially from what it was before the disease, but also the parasite: the 'cause of the disease' is perpetually being modified in the course of the disease.

But, whereas in the host this constant modification in the course of pathogenesis is obvious and incontestable, in the pathogen it can only be demonstrated macroscopically in special cases, e.g. morphologically, in the deformation of hyphal branches to haustoria (p. 324) and, physiologically, by means of chemical abilities which a pathogen develops only during its parasitic and not during its saprophytic phase. Thus *Bacterium radicola* can assimilate atmospheric nitrogen only whilst inside the host (p. 284), and ergot of rye (*Claviceps purpurea*) can form ergotamine and related alkaloids only during its parasitic phase on and in the ovaries of grasses, and not under any conditions of saprophytic culture hitherto tested. The congenial host thus evokes in the pathogens during their parasitic 'captivity' new specific processes, i.e. it induces in the pathogens certain chemical reactions.

Similar modifications of the pathogen by the host are also known in human medicine; for instance, in the course of the disease, pneumococci lose their initial marked capacity for capsule formation and for decomposition of the blood pigment, and approximate to the saprophytic streptococci of the mouth. As the illness abates, diphtheria bacilli come to resemble harmless pseudo-diphtheria bacilli and, ultimately, only the latter are found.

Since the reciprocal relationship between guest and host is continuous and, hence, the conditions under which they live are always changing, the mechanism of pathogenesis cannot be reconstructed (or only in the most rudimentary outline) by laboratory experiments isolated from this perpetual interaction of the two living partners. Thus, by means of metabolic products of the pathogen obtained by laboratory procedures, we can indeed evoke certain symptoms or symptom complexes, such as the phenomena of wilt or necrosis, but we cannot evoke the disease in its entirety.

Still less can we imitate the course of the disease, the progressive change which the clinical picture undergoes during the disease process, since these transformations depend on the fact that the action and reaction of the pathogen and the host continually progress in the course of the disease. Thus the characteristic clinical picture and the characteristic course of the disease arise only as a result of the constant mutual influence and modification of pathogen and host.

On account of this close and reciprocal interweaving of forces the infectious disease is thus more than the mere reaction of the host to the infection: it constitutes an independent process, an autonomous biological complex with its own laws; parasite and host unite after the infection has 'taken' in a communal life of a higher order which is more than their summation. An analogy to this communal life of a higher order is afforded, for example, by lichens (p. 412).

It is inherent in our scientific training that we are not yet able to regard this new life-association as such, apart from the two partners which constitute it, but try to understand it analytically as action of the pathogen and reaction of the host.

This procedure easily leads to an over-simplification of the situation. Potato wart disease is not caused by the fungus *Synchytrium endobioticum*, although in the preceding chapters we have, for the sake of brevity, repeatedly stated this to be the case: it is simply evoked by it. The fungus activates the innate proneness to wart that exists in susceptible varieties; moreover, it is not even the directly infected cells which react by the formation of tumours, but the non-infected adjacent cells. The fungus, therefore, is not the cause, but merely the agent or excitant of the wart disease; it is only possible to activate a potentiality already present. In this case the German word *Erreger* [in this book usually translated as 'pathogen'] is far superior to the French and English word 'cause'.

This function of the pathogenic agency as *Erreger* of autonomous life processes running an independent course is best exemplified in bacterial crown gall of plants, in which the disease process continues according to its own rules after the pathogen has been eliminated. In secondary tumours arising from proliferation strands at a considerable distance from the primary tumours (Fig. 67), *Bacterium tumefaciens* can in many cases no longer be demonstrated. If the bacteria-free tissues of the secondary tumours are excised and cultured *in vitro*, they retain their pathological growth-habit and later, if they are implanted in young host shoots, they develop into typical crown gall tumours (White and Braun, 1941). In a sense, therefore, the pathogen, *B. tumefaciens*, is responsible only for the initial impetus; once the crown gall reaction is under way the gall is converted into an independent biological entity, which proceeds according to its own rules in the absence of the pathogen and requires no further regulation either from the original host or from the original pathogen. We are dealing here, however, with a rare and extreme exception; as a rule, pathogen and host must remain in continued contact for the disease to pursue its course and, if the pathogen be eliminated, the disease process will be prematurely arrested.

According to the phase classification (p. 5), the occurrence of disease is the culminating point of the curve of the disease process, which is then followed by a descending limb of recovery and rehabilitation. However, since infectious diseases of plants are mostly irreversible (infection is not eliminated but only localized; p. 290), they generally lack the descending limb leading to recovery; consequently, the disease itself is usually the termination of the disease curve and, therewith, in the main, of this book.

In the present chapter some general considerations will be discussed in a first section, and the morphologic-anatomical and physiological manifestations of disease will then be dealt with in two further sections.

§ 1. General Considerations

In this section we shall deal with the following problems:

1. Disease with and without symptoms.
2. The extent of infection and the extent of disease (p. 442).
3. The relative importance of pathogen and host in symptom formation (p. 443).
4. The influence of the environment on the clinical picture and the course of the disease (p. 447).

1. *Disease with and without Symptoms*

The question as to when a disease begins will receive a different answer according to the point of view of the observer. Biologically it starts as soon as the first cellular defence reactions set in, but it only becomes manifest clinically when the reactions of the host become visible; in everyday usage, therefore, 'disease' is a clinical and not a biological conception.

The manifest reactions of the host are called the symptoms of the disease. Thus, if infection be the pre-condition of infectious disease, and the incubation period its preparation, then the disease symptoms are the expression of the resulting disease. They combine in an ordered sequence and definite intensity to form the clinical picture and course of the disease. For purposes of differential diagnosis the disease symptoms are standardized (symptomatology). According to the regularity of their appearance they may be classified as typical or as atypical symptoms, and according to their importance as major or as minor or general symptoms; for instance, in bunt of wheat the smutty disorganization of the interior of the grain is a major symptom, whereas the slightly stunted growth and the faint chlorosis of the leaves are minor or general symptoms.

Further, the disturbances to which the infection gives rise affect the course of the disease more or less markedly; thus, the first disease symptoms which appear during the incubation period are sometimes very noticeable and are then contrasted as primary effects with the later secondary symptoms (Fig. 224). Finally, a rapid post-mortem browning of the invaded tissues is common to most infectious diseases of plants.

Because they are abstract ideas the 'clinical picture' and the 'disease' are not objective entities but scientific concepts whose meaning may be controversial. In reality there are no 'diseases' but only diseased plants or there are as many diseases as there are diseased plants.

If an infection evokes no manifest reactions it is termed inapparent or symptomless and the individuals affected are known as carriers (*Keim-träger*). The cause of the non-appearance may be due to a lack of pathogenicity in the agent (e.g. Z-virus of potatoes, *Chaetomium* infection of *Lolium*; p. 108), or in an inherited or acquired tolerance in the host (e.g. ring mosaic of tobacco; p. 345). Naturally it is often difficult to distinguish between a very mild attack and a symptomless disease or symptomless infection. The expression 'symptomless disease' may itself appear to be

a contradiction, yet it is biologically apt since, biologically, the disease begins with the onset of the non-manifest cellular defence reactions (p. 441).

On this basis also, 'symptomless disease' may be differentiated from 'symptomless infection'; if cellular defence reactions are demonstrable symptomless disease is present, if they are not demonstrable there is a symptomless infection.

If previously visible symptoms temporarily disappear under certain external influences (e.g. in mosaic and similar viroses), they become masked; with the return of the plants to the original environmental conditions the symptoms likewise return; they are, therefore, reversible.

Occasionally, symptomless infections are also described as latent, quiescent, or dormant, but it is preferable to restrict this conception to those infections which are temporarily symptomless but subsequently break out. In this sense there are two forms of latency:

1. Latency as a phase of an infectious disease: here incubation is a symptomless precursor of the disease proper.
2. Latency as an independent process: an infection of bunt or loose smut on a wheat plant is latent during the early stages of growth of the host since it induces no manifest symptoms, these appearing first at the time of flowering. If a disease which has been latent for a time breaks out again, one speaks of a recrudescence.

The difference between non-appearance and latency emerges at once from the examples of Z-virus and bunt. An infection of potato plants with the Z-virus is non-apparent because it is unable by itself to become apparent, it cannot produce symptoms. On the other hand, a bunt infection of wheat is latent because it is only temporarily non-apparent and will later break out and become manifest, thus, in fact, representing a dormant catastrophe for the host.

Because of the differently constituted reaction norms of their objects the conception of latency is more restricted in plant pathology than in human medicine. The plant pathologist means exclusively the absence of gross, objectively visible disease phenomena, whereas the physician includes the subjective general condition of the patient, e.g. 'feeling ill': in man the latency period can terminate with the onset of subjective feelings of being unwell long before objective disturbances become manifest.

2. The Extent of Infection and the Extent of Disease

With regard to the spatial relations between infection and disease in infectious diseases of plants there are three possibilities:

1. The site of the pathogenic effect may coincide with the region of colonization of the disease agent.
2. A local infection may induce a systemic disease.
3. A systemic disease may manifest itself purely locally.

The first possibility is the one most commonly realized in the plant world since, as a rule, most pathogens affect only those tissues which they have

directly colonized. Therefore, in infectious diseases of plants, local disease symptoms usually correspond to a localized infection and systemic diseases to a generalized infection (p. 68). Plants do not usually react as wholes to a local infection and do not produce systemic symptoms, such as, for instance, a rise in temperature of the whole body. Even the cellular defence reactions discussed on page 281 prove to be localized.

The second possibility, systemic disease as the sequel to a local infection, is but seldom realized in infectious diseases of plants. It occurs only with certain toxin-forming pathogens (p. 242), e.g. the *Valsa* disease of stone fruit trees, the honey fungus, and some wilt-fusarioses (p. 243). The local disease focus brings about, as a distant effect, a poisoning of the whole organism, which results in sudden death immediately after the resumption of spring growth or with the onset of summer temperatures.

This particular disease course was well named 'apoplexy' in the *Valsa* disease of apricots (Défago, 1935). An insidious infection of the toxin-forming pathogen proliferates year after year in the trunk but is held in equilibrium by the defensive powers of the tree. If the tree expends its reserves in a renewal of growth, it may suddenly become unable to cope with the toxic effects, its foliage wilts within a day, and the tree succumbs. A classical example in human medicine is tetanus, in which the entire system also reacts to the local infection.

The third possibility, systemic infection with subsequent organ manifestation, also seldom occurs in infectious diseases of plants but is present in the case of a few organotropic and histotropic pathogens (p. 77). In some smut and stripe diseases of cereals (Fig. 98) the mycelium permeates the whole organism but induces manifest disease symptoms only in certain organs, e.g. the ears.

In potato leaf roll disease and curly top of sugar beet the viruses inundate the whole plant but cause only a localized cell necrosis, i.e. in the sieve tubes and their companion cells (phloem necrosis; Fig. 281). The causes of these specific organ manifestations are still unknown.

Classical illustrations from human medicine are pneumonia and typhoid, whose pathogens first initiate a systemic disease of the whole body (although for a few hours or days only), diffusion being effected through the blood stream, and are later relegated to specific organs of lower resistance.

3. *The Relative Importance of Pathogen and Host in Symptom Formation*

The relative importance of pathogen and host in symptom formation and in the course of disease may vary in one and the same host according to the symptom group and the organ group, and according to the environmental conditions. Thus, *Fusarium* disease of cereals manifests itself in seedlings as snow mould; in this syndrome the effect of the pathogen is dominant. In later stages of development of the host the disease manifests itself as foot rot (diseases of the haulm bases, &c.) and

as ear fusariosis, e.g. grain sterility; here the host reaction predominates. However, all these syndromes are manifestations of the same pathogen and the same disease but show varying degrees of dominance of pathogen and host. An infection, therefore, can manifest itself differently in the various organs of the same host plant.

The relative importance of pathogen and host in symptom formation can differ in one and the same organ according to the severity of the infection. *Bacillus atrosepticus* (potato black leg; Fig. 298), in mass infec-



FIG. 281. Phloem necrosis (dark tissue) of sugar beet due to curly top virus. Left: transverse section. Right: longitudinal section. $\times 1.5$.
(After Carsner and Stahl, 1924.)

tion, gives rise to the familiar necrosis of the cells and dissolution (wet rot) of the tubers; thus, in mass infection it takes the lead in the disease process. Conversely, in a mild attack, it merely stimulates the tuber tissue to hypertrophy, so that the host dominates the emergent clinical picture.

Even greater is the range shown by the 'effects of the pathogen on the host' and the 'reactions of the host' when different pathogens combine with different host species. In extreme cases of this kind two possibilities are open: (1) one and the same pathogen evokes different syndromes in different host species, and (2) different pathogens produce a similar or identical syndrome in one and the same host.

1. One and the same pathogen evokes different syndromes in different host species. *Corticium vagum*, discussed on page 179, may serve as an example; it is an extremely polyphagous fungus, mostly referred to in agricultural literature as *Rhizoctonia solani* (Braun, 1930).

In the first place it gives rise to different syndromes in one and the same

host, e.g. in potato plants it gives rise to stunting as a general symptom, but also to foot rot of the stem bases, necrotic lesions (stem canker) in the roots and stolons, and black scurf on the tubers.

Then it causes quite different diseases in different hosts, such as damping-off (*Wurzelbrand*) of seedlings in conifer plantations, a stem rot of carnations, beans, tomatoes, &c., a dry rot of sugar beets, a leaf spot disease (necrosis) of the leaves of cabbage, lettuce, &c., a dry fruit rot of tomatoes and bean pods, a brown rot of strawberries, and so forth.

All these diseases have the same pathogen in spite of their diverse appearance and their varied courses. They are, therefore, combined etio-logically, parasitologically, in one group according to their pathogen and termed *Rhizoctonia* diseases. The same applies to the fusarioses caused by *Fusarium* spp., verticillioses due to *Verticillium albo-atrum* and related species, and so on.

In the first place, the identity of the pathogen is important epidemio-logically in relation to crop rotation. Thus, in horticulture, the *Rhizoctonia* disease of beans can pass over in the following year to tomatoes, and later still to cabbage, &c., inducing on each of these crops diseases differing in appearance whose basic identity may not be immediately obvious to the grower.

The identity is also important parasitologically: a variety of clinical pictures does not necessarily imply a variety of pathogens. Hence reliable diagnosis must be based on the precise determination of the pathogen.

Finally, the identity is important biologically. The various forms of disease evoked by one and the same pathogen, *Rhizoctonia solani*, in the different hosts and organs constitute to some extent an expression of the specific capacity of a given host's tissues to react, i.e. their reaction potential. In reality, however, the problem must be very complicated. Indeed, in all these situations the pathogen, *R. solani*, is only juridically 'the same', systematically but not biologically the same individual, because every reaction of the host stimulates it to counter-reactions (p. 438), evokes new capacities, &c. The pathogen, therefore, is not a constant, but within the range of its innate zone of variation is ever different, different in each host and in each organ. Hence, the share of the pathogen in the differential symptom production may well be larger than a first impression would lead one to expect.

2. Different pathogens evoke the same syndrome in one and the same host (so-called 'phenotype-copies': the same clinical picture with different causes). In view of the high specificity of the pathogens and the susceptibility of the hosts one would expect a corresponding specificity of the reactions induced in the hosts. However, as a rule, this high specificity applies only to the cellular defence reactions, not to the manifest signs of injury, the actual clinical disease symptoms. Thus, it is only the defence and not the manifestation of the disease that is differentiated and highly specific. Here, the plant's possibilities of reacting are extremely limited by comparison with the great number of specialized disease agents.

The various pathological symptoms, discolorations, swellings, necroses, &c., if isolated from their context are not, in themselves, diagnostic of any given disease, but recur in all diseases—like pain, raised temperature, and quickened pulse in human medicine. It is not, therefore, the individual symptoms that are diagnostic, but the combination of symptoms, the manner of their association, their spatial and temporal relationships.

But, in reality, even these possibilities of combination are soon exhausted and, thus, given symptom complexes or symptom groups recur in the most varied hosts and with divers pathogens. Hence, in the common terminology of agriculture, forestry, and phytopathology, a number of characteristic disease syndromes have been picked out and given special names irrespective of the pathogen and host (Appel and Westerdijk, 1919; Rivera, 1942).

These include the following. Rots (Fig. 271): tissue destruction, usually accompanied in fruits by softening and discoloration of the flesh and deterioration of the flavour, and in wood by pigmentation and weakening of the cellular framework; subdivided into wet, dry, white, red, blue, green, brown rot, &c.

Spot diseases (Fig. 215): localized disease and discoloration of some tissue layers; subdivided for instance into dry spots, where the affected tissues die and become leathery or brittle, and anthracnose (*Brenner*), in which only the deeper-lying tissues are invaded and die, so that the peripheral layers, the epidermis or the cortex, sink in; a special form in seedlings is termed black leg.

Scabs (Fig. 88): a crusting of the injured skin, involving in fruits, for example, the suberization of the hypodermal cells which, therefore, cannot keep pace with the growth of the young fruits and hence rupture or give rise to distortions and deformations; subdivided into superficial, deep, pustular, tumulate, powdery, &c.

Wilt diseases (Fig. 244): constriction diseases (Fig. 286), canker (Fig. 187), &c. At the end of this series of non-specific clinical pictures stands the equally non-specific lethal conclusion, necrobiosis.

All these diseases are defined purely by their symptoms and appearance and are, therefore, completely heterogeneous in respect of their etiology. Ray fungi (*Actinomyces* spp.), Archimycetes (*Spongospora subterranea*), Ascomycetes (*Endostigme* spp.), and Basidiomycetes (*Corticium vagum*) can all evoke scab-like diseases. Plant cankers can be produced by bacteria (*Bacterium tumefaciens*, *Bacillus Savastanoi*), Archimycetes (*Synchytrium endobioticum*), Ascomycetes (*Nectria galligena*, *Dasyscypha Willkommii*, &c.), Basidiomycetes (*Melampsorella caryophyllacearum*), as well as purely physiogenically by frost action on dormant buds. Hence, the fact that clinical pictures are identical should not lead to the assumption that the pathogens are identical.

On account of this marked convergence of clinical pictures in etio-logically heterogeneous infectious diseases (e.g. the innumerable leaf spot diseases), clinical diagnosis, which seeks to infer the causes of disease

indirectly from the mode of reaction of the infected organism, is attended with greater uncertainty in phytopathology than in human medicine. Only etiological diagnosis is reliable, i.e. the direct microbiological (parasitological) demonstration of the pathogen or of the specific products of reaction. This demonstration is facilitated in the mycoses by the fact that the pathogen often begins to reproduce on the diseased tissues quite soon after incubation, and may readily be identified by its spore forms.

In the same way no direct relation exists between the clinical picture or the parasitological characters of the pathogens, and their phylogeny, i.e. their evolutionary status. Biologically inferior pathogens disorganize and kill their hosts; biologically superior ones compromise with their hosts, allow them to continue their vital functions, and merely stimulate or incite them to special activities (tumours, increased respiration rate, &c.).

However, the biologically inferior grade of parasitism is exhibited by pathogens of both phylogenetically low status (*Pythium de Baryanum*) and phylogenetically high status (*Corticium vagum*); similarly, the biologically superior form of parasitism is exhibited both by lower types (*Synchytrium endobioticum*: potato wart) and higher types (*Melampsorella caryophyllacearum*: witches' broom of silver fir). The rust and smut fungi, however, do not induce rotting of their host plants.

4. *The Influence of the Environment on the Clinical Picture and the Course of the Disease*

The environment spontaneously influences not only the vitality of the pathogen (p. 192) and the disease proneness of the host (p. 379), but also the result of their interaction, the disease itself (phenotypic variations in disease manifestation). The phenotypic changes in the duration of the incubation period (p. 418) also support this view. Yet it is far from easy to delimit these various spheres of influence. Reaction type in rust diseases may be altered by host nutrition and atmospheric temperature; this effect is due to modifications in the host, in the pathogen, and in their communal life. In the absence of factual data, however, arguments of this kind tend to over-subtlety.

However, the influence of the environment on symptom formation is of practical importance in many viroses. Thus, under favourable cultural conditions, chronic infections often undergo a diminution or a masking of their symptomatic expression, and this makes it hard to estimate how far diffuse diseases and yield reductions, e.g. in a given potato variety, are to be attributed to physiological degeneration and how far to chronic virus infection. A decision can only be made by re-isolating the virus in test plants.

§ 2. *Morphological-Anatomical Manifestations of Disease*

The presence of disease can show itself in disturbances of organization (form) and in disturbances of function (performance). It is clear that form and function are linked together but, in order to give a description at all, we

are compelled to isolate the several components of the host and treat them separately.

The morphological, histological, and cellular components of the host affected by disturbances in equilibrium form the subject matter of pathological morphology (teratology) and pathological anatomy.

The physiological components of the host affected by disturbances in equilibrium form the subject matter of pathological physiology. This division of symptoms corresponds to the academic arrangement of the curriculum.



FIG. 282. The influence of *Gibberella Fujikuroi* on the growth in height of rice seedlings. 1 Weak seedlings whose development has been inhibited by infection. 2 Controls; non-infected seedlings. 3 Vigorous seedlings whose growth in height is excessive because of infection (bakanaë effect). (After Seto, 1935.)

The present section is concerned with the morphologic-anatomical manifestations of functional disturbance in the host: (1) disturbances in growth and form of the host (teratology), and (2) disturbances in its anatomic-histological structure (pathological anatomy). A detailed description of these disturbances, in so far as they are induced by fungi, is given by Fischer and Gäumann (1929).

1. Disturbances in Growth and Form of the Host (Teratology)

Malformations, i.e. disturbances in the growth and form of the host, are termed morphoses. When, as in infectious disease, they are provoked by living agencies (not by heat, frost, &c.), they are called biomorphoses.

This formative influence on the host may be manifested, in the first place, in systemic symptoms, e.g. in an increase or in an arrest of the total growth of the attacked individuals, but the boundaries between typical, atypical, and pathological growth are often hard to discern.

A general stimulatory effect may be exerted by certain *Fusaria*, e.g. *Gibberella Fujikuroi* and *Fusarium moniliforme*, on vigorous young plants

of rice, oats, maize, &c. Weak individuals are arrested in their development by these fungi and lag behind (Fig. 282 1), whereas robust plants grow taller (Fig. 282 3), so that they become rather etiolated and are conspicuous in the field by their height (bakanaë disease of rice). Moreover, the production of dry substance by infected individuals is significantly higher, especially at soil temperatures above 22° C. (Fig. 283; Table LXXXII).

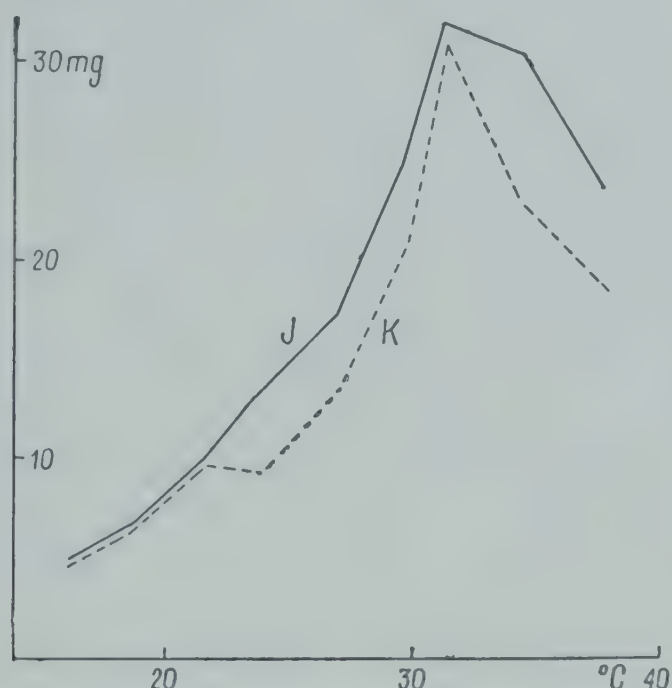


FIG. 283. The stimulating effect of *Fusarium moniliforme* on the production of dry matter by young rice plants. Abscissae: soil temperature. Ordinates: average yield of dry substance per plant. Curve J: infected rows. Curve K: controls. (After de Haan, 1937.)

TABLE LXXXII

The bakanaë effect of Fusarium moniliforme on young rice plants of the variety Si Landjah. (After de Haan, 1937)

Soil temperature ° C.	Average height of plants		Average dry weight of plants	
	infected row	control row	infected row	control row
	mm.	mm.	mg.	mg.
16	74 ± 1.3	71 ± 1.2	4.8 ± 0.2	4.6 ± 0.3
18.4	94 ± 2.1	87 ± 2.3	6.6 ± 0.5	6.2 ± 0.2
21.7	125 ± 1.7	97 ± 1.5	10.0 ± 0.4	9.8 ± 0.5
23.8	142 ± 1.8	134 ± 1.4	13.5 ± 0.4	9.5 ± 0.3
26.9	160 ± 2.4	151 ± 1.8	17.5 ± 0.3	13.7 ± 0.4
29.7	188 ± 1.1	177 ± 2.1	25.1 ± 0.6	20.7 ± 0.5
31.2	194 ± 5.6	189 ± 3.2	32.3 ± 1.1	31.2 ± 0.8
34.3	171 ± 3.0	157 ± 2.4	30.7 ± 1.5	23.3 ± 1.0
37.7	152 ± 3.8	140 ± 3.3	24.0 ± 1.2	19.0 ± 1.1

The active principle, gibberellin, is still effective at a dilution of 1:10⁻⁷ (Yabuta and Hayashi, 1939); this corresponds to 2 µg. per seedling or G g

10 mg. per kg. of living plant tissue. A second substance, fusaric acid, induces similar elongation at a dilution of $1:10^{-6}$ (Yabuta and Hayashi, 1940).

More frequent than generalized stimulation is general arrest of growth or stunting of the host plant. Plants affected by bunt are smaller throughout than healthy ones and have a lower productivity measured in weight per 1,000 grains and in straw yield (Tables LXXXIII and LXXXIV); furthermore, their root system is weakened, so that as a frequent secondary effect of latent infection there is a higher incidence of winter killing of infected plants (Table LXXXV). Other effects, however, such as the influence on tillering, vary from one variety to another (and perhaps also from one strain of *Tilletia* to another); thus (Table LXXXIII) tillering is retarded in winter wheat and (Table LXXXIV) stimulated in spring wheat, but possibly indirect changes are also involved (e.g. increased susceptibility to frost; Tapke, 1929).

TABLE LXXXIII

General morphological symptoms of bunted winter wheat, var. Hohenheimer 77 bearded. (After Gieseke, 1929)

<i>Character</i>	<i>Infected</i>	<i>Controls</i>
Length of longest haulms (cm.) .	106.2	120.3
No. of haulms per plant . .	3.7	4.4
Length of ear (cm.) . . .	7.4	8.0
No. of spikelets . . .	23.5	23.8
Weight per 1,000 grains . .	50.3	54.0

TABLE LXXXIV

General morphological symptoms of bunted spring wheat, var. Hen Gymro. (After Sampson and Davies, 1927)

<i>Character</i>	<i>Infected</i>	<i>Controls</i>
Average length of haulms (cm.) .	80.2	98.8
No. of haulms per plant . .	8.9	7.7
Length of ear (cm.) . . .	9.6	10.6
Straw yield per plant in g. . .	11.9	16.9

These general inhibitory results of infection may be dependent, for example, on direct withdrawal of nourishment by the pathogen, or on functional disturbance of either nutritional or growth substance metabolism.

However, a given pathogen need not be only inhibitory or only stimulatory in effect, but may unite inhibitory effects on one factor complex with stimulation of another. Thus, in *Panicum* smut inhibition of growth in length is combined with promotion of leaf development (Fig. 284); the plants with latent infection are more compact and bushy.

In extreme cases arrest of growth leads to dwarfing (nanism; Fig. 285); such an overall effect is naturally non-specific and may equally well be due to foot rot (*Rhizoctonia solani*), to black leg (*Bacillus atrosepticus*), or to a mixed virosis.

TABLE LXXXV

Winter killing (in % of growing plants) of wheat infected with *Tilletia tritici* (bunt), and of barley infected with *Helminthosporium gramineum* (leaf stripe). (After Zade, 1932)

Variety	Infected	Not infected
	%	%
Heils Dickkopf wheat	34.0	9.0
Eckendorfer Mammoth winter barley	31.6	14.0
Friedrichswerther winter barley .	47.6	26.8
Groninger winter barley	54.4	23.2
Strengs winter barley	56.4	28.4
Kalkreuther Universal winter barley .	62.0	32.0

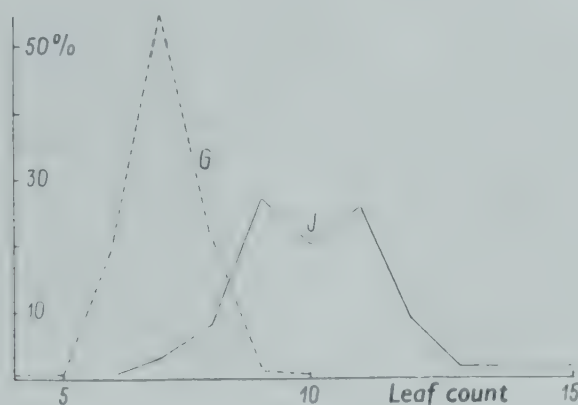


FIG. 284. The number of leaves, in a pure line *Panicum*, of plants infected and not infected by the smut, *Ustilago panici-miliacei*. Abscissae: number of leaves. Ordinates: percentage number of individuals which show the leaf count indicated. Curve G: healthy plants. Curve J: infected plants. (After Buchheim, 1930.)

Unlike these general symptoms, the morphogenic disturbances in infectious diseases of plants are usually localized. Inhibitory factors (generally associated with parasitogenic disintegration of the tissues) lead to atrophy (*Gewebebeschwind*), and stimulatory influences lead to hypertrophy.

An example of local atrophy, associated with local necrosis, is the constriction or girdle disease of conifers (Fig. 286). In leaf scorch of cherry (*Gnomonia erythrostoma*) the formation of abscission tissue at the base of the petioles in autumn is suppressed, so that the drying leaves remain suspended on the trees throughout the winter.

The type and extent of local hypertrophy depend (apart from the pathogen) on the reactivity of the affected tissues, particularly on their capacity for regeneration, i.e. for induced meristematic activity. The newly forming tissues almost always undergo some physiological modification.

Thus histogenic demarcations (see p. 330) are to a certain extent 'internal hypertrophies'. We shall consider briefly four groups of hypertrophic parasitogenic morphoses: gall formations, tumours, witches' brooms, and the activation of rudimentary sexual organs.

In the simplest cases, i.e. in gall formations, intercellular organization is disturbed only in the case of single cells or parts of tissues: cells or cell groups which appear to have completed their differentiation are freed from restriction by the influence of the pathogen, swell, divide, and begin to develop independently as if they were a special organ. Such morphoses,



FIG. 285. Potato plants, var. Up-to-date. Left: infected with *Verticillium albo-atrum*, the pathogen of a tracheomycosis. Right: control. (After Donandt, 1932.)

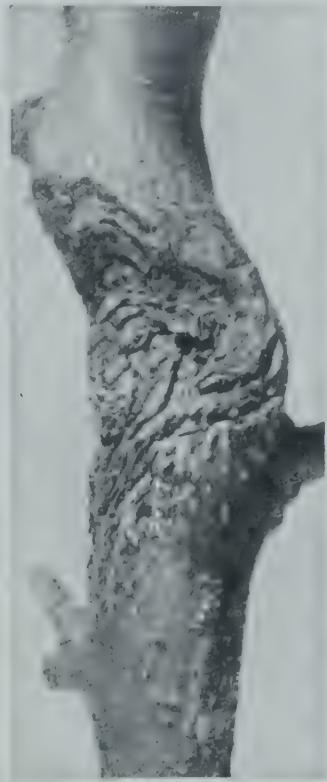
generally fungal galls or mycocecidiae, range from simple swellings, thickenings, callosities, &c., to smut boils as large as a child's head, as in maize (Fig. 144). They usually contain more water than healthy tissues: thus healthy leaves of cranberry (*Vaccinium Vitis-idaea*) have a dry content of 43.6% compared with 21% in those attacked by *Exobasidium vaccinii* (Küster, 1911).

A special and, in its way, characteristic combination of local gall-like swellings and physiological modifications occurs in a certain group of canker diseases. It is known that powdery mildew of oak (*Microsphaera alphitoides*) and of vine (*Uncinula necator*) delay the maturation of the wood to full frost hardiness, so that the diseased branches are easily frosted in autumn. This softening effect in larch canker (Fig. 275) and also in apple canker (Fig. 287) is additional to the general parasitological syndrome and is superimposed upon it, so that there arises in larch canker, as illustrated on page 432, a combination of local hypertrophies associated with parasitogenic necroses and exogenous frost injury.

In more complex cases the galls develop adventitious vascular bundles, &c., and to some extent lead an independent life; they are then known as tumours (e.g. the root nodules of leguminous plants, Fig. 178; wart disease of potato, Fig. 187; and bacterial canker, Fig. 288). They originate generally from the cells of the cambial or cortical parenchyma, are analogous to the connective tissue cells of animals and, therefore, correspond histologically to the sarcomata of human pathology.



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FIG. 286. Local atrophy (girdling disease caused by *Phomopsis pseudotsugae*) of the main stem of a 4-year old Douglas fir. Original. Nat. size. (Photograph Malcolm Wilson.)

FIG. 287. Local hypertrophy associated with local necroses and frost injury (twig canker) of apple, var. Landsberger Reinette, caused by *Nectria galligena*. Original. $\times \frac{1}{2}$. (Photograph R. Maag.)

On account of their supposed analogy with malignant growths (carcinoma) of man, tumours (canker or crown gall) evoked by *Bacterium tumefaciens* have acquired a special biological significance. They arise spontaneously following wound infections on the root collar or the roots of all kinds of cultivated and wild plants, generally in the form of tuberous, strumose, or cauliflower-like swellings which occasionally may reach the size of a child's head. Experimentally, they may be induced by needle inoculation of both stems and leaf veins, and the plant may respond with a vigorous formation of roots in addition to the actual tumour (Fig. 289).

It was noted earlier (p. 65 and p. 440) that these tumours, like malignant growths, may, on occasion, give rise to metastases and that they are transplantable. But, corresponding to the different form of organization of the plant body with its rigid scaffolding of cellulose walls, the mechanism

of metastasis is different. In man it is freely mobile cancer cells which migrate through the lymphatic system, &c., and initiate new foci; in plants it is compact cell strands which grow out from the cancer focus and give rise to a new focus at some distance (Fig. 67).

A similar partial coincidence and partial divergence between animal-human cancers and plant cancers exists in respect of etiology. Agreement between plants and man obtains in the primary conditions of cancer: in



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FIG. 288. Crown gall of beet caused by *Bacterium tumefaciens*. Approx. $\times \frac{1}{4}$. (After Stapp, 1928.)

FIG. 289. Tumour and root production on a stem of *Impatiens Balsamina* evoked by *Bacterium tumefaciens*. $\times \frac{3}{4}$. (After Brown, 1929.)

both realms the individuals must possess an initial, latent proneness to cancer. But the agency which evokes cancer is different. In plant tumours it is, in the present case, certainly a micro-organism, *Bacterium tumefaciens* (or, as has already been suggested, a cohabiting virus), whereas in animal or human cancer, vegetative mutations, chemical stimuli, and, in sarcoma, pathogenic viruses, are mainly involved.

It is true that plants can also respond to chemical stimuli (including extracts of *B. tumefaciens* itself; Boivin *et al.*, 1937) by tissue growths and new formations, but here substances different from those in the animal kingdom are effective. Among the fifty or more carcinogenic substances of animals, aromatic hydrocarbons (e.g. 3:4-benzpyrene), azo dyes (e.g. o-amido-azotoluene), and the dyes of the styrylquinoline group are especially active. So far as they have been tested, individual substances from these groups, e.g. the higher temperature fractions (b.p. 330–360° C.) of coal and beech-wood tar, as well as 1:2-benzpyrene, are also effective

in plants, but only as general stimulants, not as carcinogenic agents. Thus, appropriate treatment with these substances stimulates the production of numerous adventitious roots in *Solanum Lycopersicum* and *Nicotiana suaveolens* (Kisser and Lindenberg, 1940). Hence, the possibility of chemical stimulation of tissues is present in both plants and animals, but the point of attack in the cells or the reaction of the organisms concerned are different: the plant responds non-specifically with root or callus formation, whereas the animal responds specifically with cancer formation.

In plants it is mainly growth substances which are carcinogenic, especially indolylacetic acid (heterauxin), although they elicit no response in the animal body; in beans heterauxin gives rise to growths which, histologically, are very similar to bacterial tumours (Kraus *et al.*, 1936; Hamner and Kraus, 1937).

Because of this carcinogenic effect of growth substances, because *Bacterium tumefaciens* produces the same growth substances in artificial culture, and because bacterial galls of young tomatoes are much richer than healthy tissues in growth substances (Locke *et al.*, 1938; Link and Eggers, 1941), an attempt has been made to explain the mechanism of tumour formation quite simply on a growth substance basis: the growth substances emanating from the bacterium evoke the tumour. This interpretation is supported by the observation that extracts of *B. tumefaciens* cultures and of bacterial tumours give rise, on suitable plants, to growths which appear more or less similar to gall tissue. However, this explanation has proved to be too simple.

Thus, growth substance production by *B. tumefaciens* (at least in artificial media) reaches at most a concentration of 125 $\mu\text{g.}$ per litre (Locke *et al.*, 1939), whereas the artificial production of galls generally needs concentrations of 10–30 million $\mu\text{g.}$ per litre, i.e. some hundred thousand times higher (Riker *et al.*, 1941). The higher growth substance content of the tumour tissues probably depends, therefore, not on the pathogenic agent but on a stimulation of growth substance production induced by the host.

Moreover, the host range of *B. tumefaciens* does not coincide with the pathogenic range of indolylacetic acid. There are plants that produce tumours under the influence of *B. tumefaciens* but not when treated with indolylacetic acid, and, conversely, there are some that respond to indolylacetic acid but are not susceptible to *B. tumefaciens* (Riker, 1940).

Finally, the same growth substances can also be produced in artificial nutrient media by bacteria, e.g. *Bacillus radiobacter*, which do not give rise to any tumours (Locke *et al.*, 1939). The causes of morphogenic and carcinogenic effects are evidently diverse in origin (p. 440); but tumour initiation in plants quickly leads to a disturbance of growth substance metabolism, whereas animal cancers lead rather to disturbances of the proteolytic ferment system (δ -amino-acid content of tumours, &c.).

As in etiology so also in histology, the malignant growths of animals and man and those of plants show points in common and points of difference. Similarity obtains, for example, in respect of their cellular pathology (Stapp, 1927; Riker and Berge, 1935). The point of origin of the growth is always in cells directly affected by the stimulus, which begin to divide atypically (Fig. 290); in plants, usually cells of the cambium, cortex, pith,

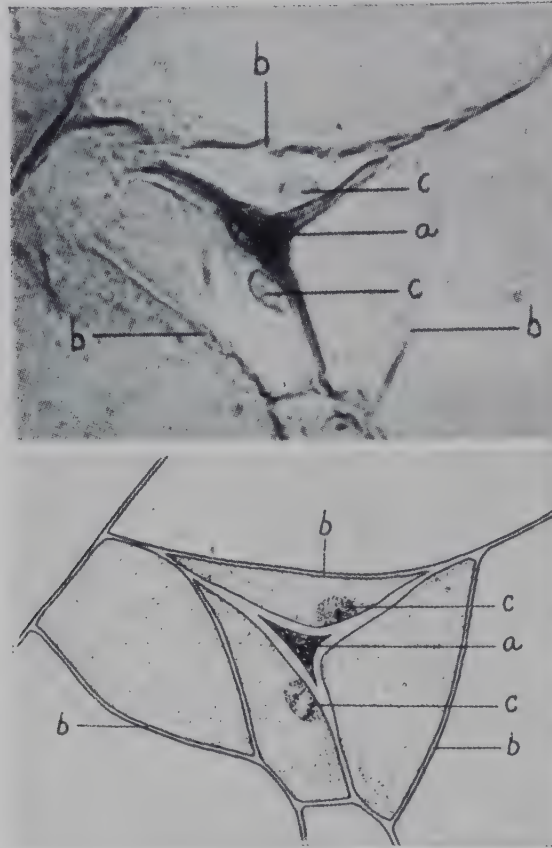


FIG. 290. A section through the starting-point of a bacterial canker of tomato 8 days after infection by *Bacterium tumefaciens*. Above, photomicrograph; below, diagrammatic representation. The bacterium-containing intercellular cavity *a* has stimulated nuclei *c* of the neighbouring host cells and initiated the primordia of periclinal walls *b* through atypical division. Approx. $\times 900$. (After Riker, 1923, from Riker and Berge, 1935.)

or medullary rays, more rarely of the epidermis. The young, deep-seated tumour is free to expand in the intercellular spaces and, later, appears to be able to grow by 'apposition', i.e. by the transformation of adjacent normal parenchymatous cells into tumour cells.

In the subsequent course of development, both in carcinoma and in plant tumours, a certain histological differentiation takes place. The ground tissue in plants then consists of strongly staining isodiametric or spindle-shaped cells (Fig. 291) which, as in animals, may be uninucleate or multinucleate (up to 30 nuclei). The nuclei become enlarged, stellate or fissured, &c., occasionally polyploid and capable of amitotic fragmentation. Between the cells of the ground tissue, vascular bundles develop irregularly (Fig. 292), and are generally unconnected with the vessels of the organ

in which the tumour arose, although in deep-seated infections the tumours may extend far into the host tissues.

Thus there is fundamental agreement between plant and animal tumours in the functionless development of the growths, in their atypical organization (loss of polarity), in their poor capacity for differentiation, in the insufficient vascularization of the gall tissue, in their autonomous behaviour after transplantation as if they were themselves parasitic, and in the degenerative changes within the cells.

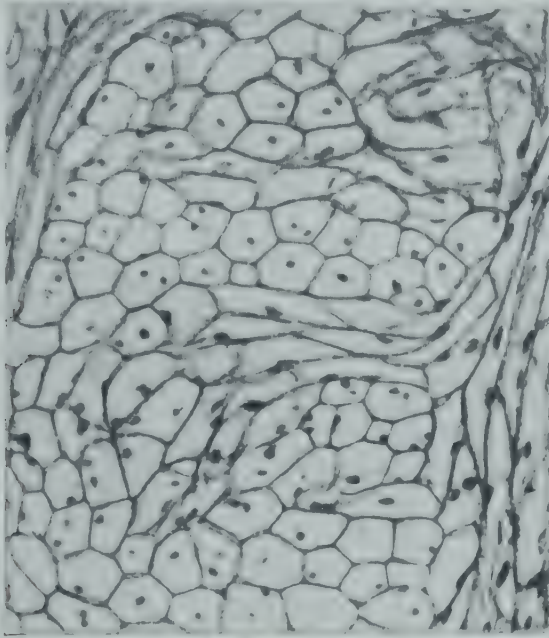


FIG. 291. Parenchyma of an experimentally induced bacterial tumour on *Chrysanthemum indicum*. The cells, some of which are binucleate, are dividing at random. Approx. $\times 400$.
(After E. F. Smith *et al.*, 1912.)

On the other hand, the histological relationship of the tumour to adjacent tissues is markedly different: in plant tumours the specific character of malignancy is absent, i.e. there is no destruction of neighbouring tissues by infiltration or expansion into them.

As in respect of pathology and histology, so also in respect of physiological metabolism there are probably some similarities and some differences between animal-human and plant tumours. But in plant pathology this field has so far been little explored.

The problem of bacterial plant tumours is thus doubtless of far-reaching biological significance and, since all life is life, its investigation may give some insight into medical problems. In spite of the same name being applied to the diseases (*Krebs*) and their similarities and convergences, it should not be concluded that they are due to the same causes; they are analogues rather than causal homologues.

Among the morphoses so far discussed, galls, tumours, &c., the morphogenic stimulus affects only single cells or portions of tissue; if it affects whole buds, then 'witches' brooms' (Fig. 230) generally arise. Yet in spite of

their difference in appearance, plant galls and witches' brooms merge into each other; thus, the wart of potato (Fig. 187) is a combination of parenchymatous growths (cankorous tumour formations) and deformed shoot systems (witches' brooms).

In most cases, owing to the infection, the bud is diverted from its original goal of developing, for the good of the whole plant, an assimilatory branch with flowers and fruit, and it becomes isolated from the common

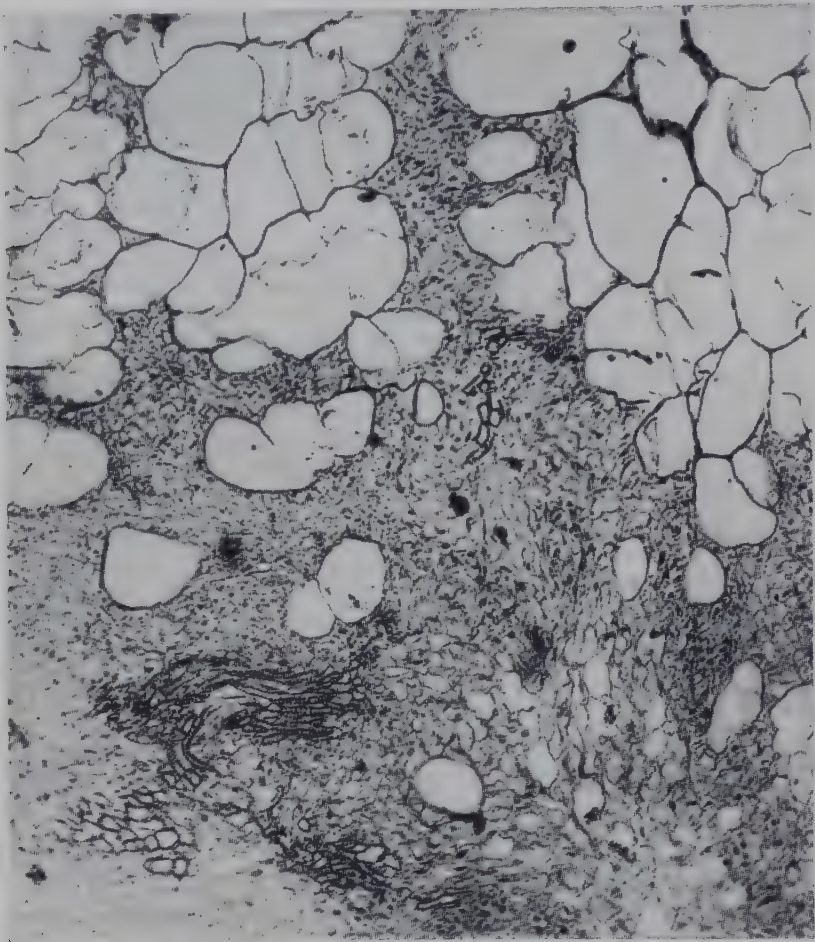


FIG. 292. Above, hypertrophic and, below, hyperplastic parenchyma, with adventitious vascular bundles (left below) of a tomato tumour evoked by *Bacterium tumefaciens*. $\times 94$. (After Riker, 1927, from Riker and Berge, 1935.)

life of the whole. The witches' broom represents an independent individual which, as a parasite, leads its own life on the parent plant: if one grafts a twig of a witches' broom of sweet cherry caused by *Taphrina cerasi* on to a healthy sweet cherry tree, it gives rise to another 'normal' witches' broom (Heinricher, 1915).

In accordance with their independent existence, witches' brooms usually tend to break away from the correlations of the parent plant. This is first manifest in their parasitogenic sterilization; as a rule witches' brooms produce no flowers.

Moreover, their independence expresses itself in changes in their tropic behaviour: instead of spreading out horizontally as in healthy plants, the branches, e.g. of the witches' broom of white pine (Fig. 230), are erect,

and the needles, as in the apical shoots of white pine, are arranged radially instead of in two rows.

Finally, the independence of the witches' broom manifests itself in an impaired periodicity. On the cherry tree brooms just mentioned, the buds open 2 weeks, and on white pine brooms 3-6 weeks, earlier than those on healthy branches (Schellenberg, 1915). This premature unfolding is obviously connected with the fact that the diseased parts, e.g. the shoots mentioned on page 452, almost never attain a true autogenous winter dormancy; since the fungal mycelia permeating them never become entirely quiescent, but only reach a state of retarded growth with increased density of protoplasm. The interaction between pathogen and host thus undergoes no interruption in winter-time but is maintained in an attenuated form. It finds expression in a heightened rate of winter respiration of the affected twigs, and also in the fact that the first-year twigs of witches' brooms are usually overtaken by winter frosts and killed back.

As the brooms undergo only an enforced, parentally determined resting period, it is possible for them to begin to bud early in November. In this case they first make use of the rich nutritive reserves stored in their tissues and thus, initially, they live by their own efforts and without assistance from the host. However, this is only possible for any length of time where carbohydrates are concerned, but not water, of which their own reserves are insufficient. If, for example, a witches' broom of white pine with a length of the healthy shoot bearing it be placed in water, then the buds of the broom become swollen, but quickly wither: the water in the broom is inadequate to support the increased vital activity and, further, the water circulation in the healthy shoot is at a standstill because of winter dormancy. If the broom be cut off closely, so that its tissues come into direct contact with the water, the buds unfold without any disturbance.

It can be said, therefore, that the winter dormancy of the witches' broom, apart from climatic conditions, is determined by the cessation of sap circulation in the tree; as soon as this recommences in the early spring, the broom can continue its growth with the help of its own stores at a time when the healthy buds of the host still remain dormant.

Finally, a special group of parasitogenic morphoses includes the activation of rudimentary sexual organs. If female individuals of the white and of the red campion (*Melandrium album*; *M. dioicum* = *M. rubrum*) are attacked by the pathogen of anther smut (*Ustilago violacea*) which forms its brandspores only in anthers, i.e. only in the male reproductive organs (p. 79), then the parasite prevents the normal arrest of stamen development, allows the small stunted primordia to reach full development to serve its own extraneous ends, and then proceeds to form its brandspores in them. The ovary, however, although externally only slightly reduced, is functionless. A similar process occurs in the gynodioecious (separate hermaphrodite and female organs occur on two different individuals) *Knautia arvensis* and *K. sylvatica* due to *Ustilago scabiosae*.

The physical bases of the morphogenic stimuli which overcome the

arrested development of the male sexual organs in the female flowers are still unknown.

2. Disturbances in the Anatomic-Histological Structure of the Host (Pathological Anatomy)

The disturbances in the anatomic-histological structure of the cells and tissues constitute, as it were, the internal symptoms of disease. Their fundamental importance has been investigated by Küster (1911, 1916, 1929, 1930) so that here only a few words are necessary. According to their nature the anatomic-histological manifestations of disease may be stimulatory, inhibitory, or disintegrative (destructive).

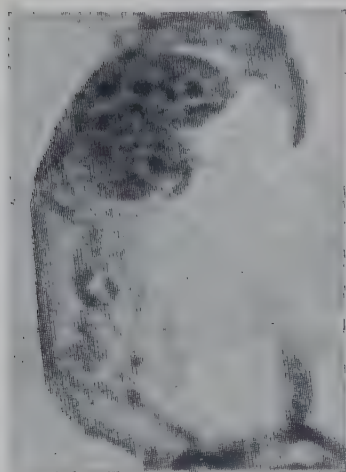


FIG. 293. x-body (intracellular inclusion) in the epidermal cell of a tobacco leaf, infected with L-strain of virus X. *a* nucleus. *b* x-body. Approx. $\times 1,500$. (After Salaman, 1938.)

Stimulation resulting in cell enlargement leads to hypertrophy of cells and tissues, whereas stimulation resulting in increased cell number, i.e. new formation by cell division, leads to hyperplasia (Fig. 292). A special group of new formations includes wound and regeneration tissues, which first bring about the closure of a wound (Fig. 212) and then rebuild the tissue which has been destroyed (restitution). As well as the cells, some of their organs, e.g. the nucleus (Fig. 6) or the chloroplasts (Fig. 294) may swell and hypertrophy, or the cell wall may thicken, &c. Finally, metaplasia (re-differentiation) may occur; thus under the influence of white blister (*Albugo candida*) the stamens and petals of crucifers may begin to develop numerous chloroplasts (greening). In the in-

infected cells of most viroses so-called x-bodies occur (Fig. 293), i.e. granular, rarely vacuolate, easily stainable inclusions of unknown function, which may show more deeply staining portions like nucleoli (Sheffield, 1931).

The inhibitory effects lead to a retardation of development in certain cells, tissues, or organs and, on occasion, may terminate in atrophy (p. 451). However, inhibitory and stimulatory effects may be combined in the one organ; thus Fig. 294 shows an hypertrophy of infected palisade and spongy parenchyma cells (hence the wrinkling of infected leaves) together with a suppression of chloroplasts (hence the light-green colour of the affected parts of the leaf).

The disintegrative effects of the pathogen are, in the most primitive instances, purely mechanical in nature; the pathogen consumes the host. *Synchytrium endobioticum* and similar intracellular parasites devour only the cell contents (cytolysis); others, e.g. *Phytophthora infestans*, in addition devour the middle-lamellae (pectins); yet others, e.g. *Trametes radiciperda* on firs, break down the cellulose framework of the cell walls, leaving the lignin behind, and in this way bring about a red rot of the wood; still other

fungi, e.g. *Stereum purpureum* on beech, dissolve out the hard parts, the lignins, instead of the cellulose, leaving among other substances the colourless cellulose (white rot). In both red and white rots the specific gravity of the wood diminishes according to the amount of rotting.

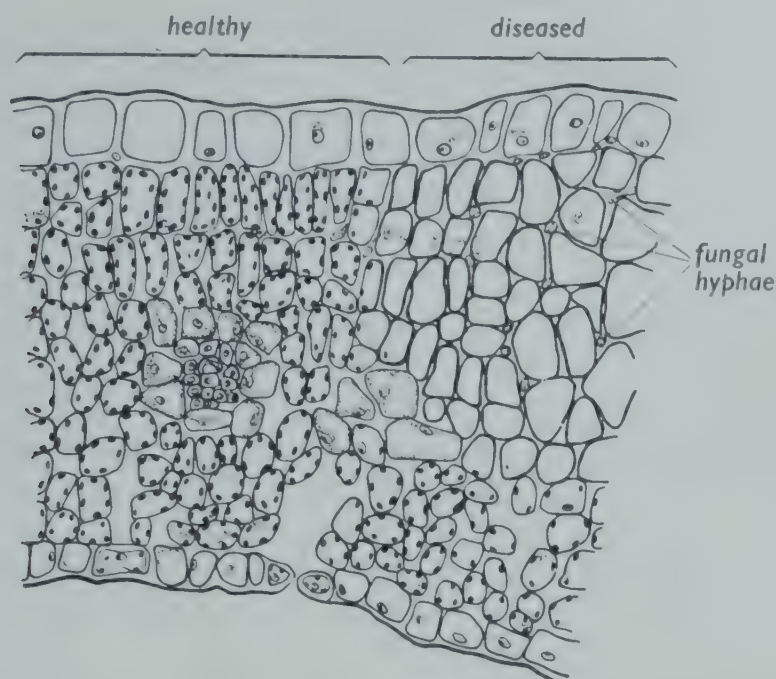


FIG. 294. A section through a peach leaf partially infected with *Taphrina deformans* (peach leaf curl). In the diseased portion the cells have hypertrophied and the chloroplasts have degenerated. Approx. $\times 300$. (After Wallace and Whetzel, 1910.)

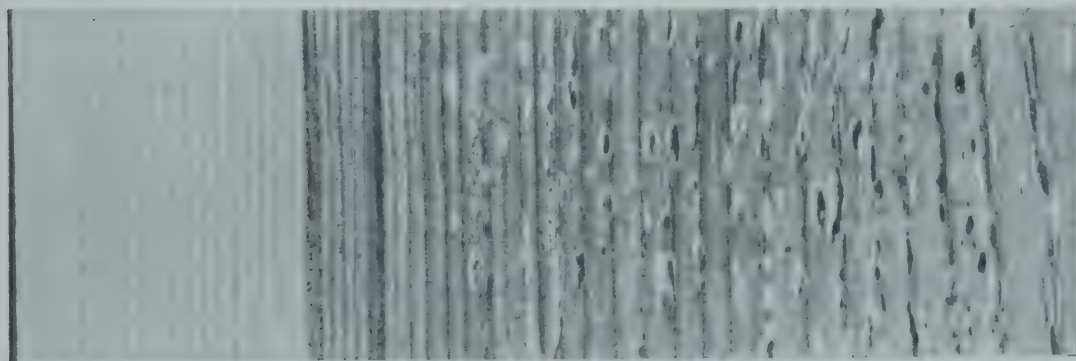


FIG. 295. Heart-wood of pine showing extensive decay caused by *Trametes pini*. In the white pockets the fungus has dissolved the middle lamellae and lignin so that only cellulose fibres remain; hence the white coloration. Nat. size. Original.

Finally, these forms of gross mechanical destruction lead to the production of actual cavities, as in the wet rot of potatoes caused by *Bacillus atrosepcticus* and related bacteria. In forest trees the corresponding syndrome (Fig. 295) is characteristically described as partridge-wood; like most syndromes it is non-specific, being caused in oak by *Stereum frustulosum*, in fir by *Trametes pini*, and in white pine by *Polyporus Hartigii*.

In more advanced cases the pathogen does not begin its work of destruction with a direct enzymatic solution of the host cells, but with a

disturbance of their functional capacity and their way of life which, however, in its turn, leads finally to death; only then does complete absorption of their nutrients take place. Thus, in such cases the pathogen no longer completely dominates the course of the disease, but this is also influenced to some extent by the reaction capacity of the host: the host is not consumed during life but after death.

These perthophytic disintegrations are usually purely local in effect; in many leaf spot diseases (Fig. 215) the invaded tissues first die, whereupon the pathogen obtains from them useful foodstuffs which enable it to produce spores (Kerling, 1928).

§ 3. Physiological Manifestations of Disease (Pathological Physiology)

The physiological reactions of the infected host may be specific or non-specific. Defence reactions are specific and these have already been dealt with on page 278 et seq., whereas disturbances of metabolism and physiologically stimulatory functions, &c., which still remain for consideration, are non-specific. The present section is limited to physiological disturbances in metabolism, because pathogenic stimulus physiology is of little significance in botany.

It may be well to repeat that metabolic physiological disturbances are only one aspect of the diseased state and that morphologic-anatomical disturbances are closely linked. Thus the same disease can manifest itself anatomically as a corrosion or decolorization of the chloroplasts, and physiologically (functionally) as an impairment of assimilatory activity. The two aspects combine to form the clinical picture of the given disease.

As in the morphologic-anatomical disturbances, so also when its physiological functions are disturbed the host attempts to restore equilibrium: impairment or disturbance of function and restitutive or regulatory activities go hand in hand. Research on the pathological physiology of metabolism has only recently been undertaken and we cannot yet trace the inter-reactions of the two processes of impairment and restitution, but that the play and interplay of these forms is not only to be found in animal-human but also in the plant organism may be presumed on the grounds of induced premunity (p. 307) and the development of acquired tolerance (p. 343). The metabolism of the host alters during the development of the new immunological capacities and its functional adaptation to infection (regulation) changes it from its previous condition; in this way it conduces to the acquisition of immunity.

In the metabolic-physiological sphere, in the narrow sense, this infection-induced struggle to establish a new biological balance only becomes evident if the functional adjustment, the inner compensation, ceases to be equal to the demands made upon it, so that very obvious disturbances arise. These tend either to a heightening of function, i.e. a stimulation, or to an inhibition of function, i.e. an impairment of efficiency.

We may assume that every infection is initially stimulatory. In human pathology this stimulus effect leads first to inflammation, followed by local infiltration. In plant pathology this characteristic primary stage is lacking, but the occurrence of the stimulatory effect may be inferred from the course of the infection itself (e.g. in the traumatotactic movement of cell nuclei; Fig. 7) and from the cellular defence reactions.

A non-specific functional general symptom of this parasitogenic stimulus effect is an acceleration of the basal metabolism of the diseased individuals or tissues. This can, for example, be detected in an increased rate of respiration, which will be discussed later.

In some leaf-inhabiting pathogens the parasitogenic stimulation becomes visible during the autumnal yellowing of the leaf. For instance, the infection foci of some rust fungi, in contrast to the yellowing of the uninfected parts of the leaf, remain surrounded by a green halo for a long time. In the same way the infection spots of sycamore mildew (*Uncinula aceris*) and tar spot (*Rhytisma acerinum*) usually show as green islands on the autumnally coloured leaves. The attacked leaf tissue does not take part in the autumnal decline in vital activity; the parasite keeps it, as it were, in a state of excitation and does not allow it to become dormant: this effect is shown in another way in witches' broom (p. 459) and larch canker (p. 432). For the same reason a smut or heavy rust attack (Murphy, 1939) reduces the winter hardiness of cereals so that they freeze more easily.

The stimulatory effect of the pathogen endures as a rule until the climax of the disease, i.e. the crisis. In some leaf diseases this shows macroscopically in the changed colour of the diseased tissues, from light green to yellowish-brown. With the crisis manifest physiological injuries to the host organism occur, which lead to impairment of function and, therefore, to a decreased productivity of the host.

The decline in both the amount and the quality of the yield which appears in most cultivated plants as a consequence of infectious disease is made up of three components: (1) the direct removal of nutrients by the pathogen, (2) the parasitogenically accelerated basal metabolism of the host, and (3) the interference with the assimilatory metabolism of the host.

1. The withdrawal of nutrients used by the pathogen for its own requirements. This can only be measured in a few cases in which the amount of material extracted, its body weight, can be ascertained, as in ergot of rye. In Fig. 296 the abscissae record the number of ergots in an ear of rye, the ordinates the average weight of the ergot yield per ear of rye as a percentage of the total weight of the ear (i.e. rye grains plus ergot sclerotia). The reciprocal relation between the two partners finds clear expression here: the greater the number of ergots per ear the greater (in spite of the general decrease in size of individual ergots) is their total weight, and the smaller the amount of nutrient still available for the production of rye grains. With more than twenty *Claviceps* infections per ear, only about 20% of the reserve material transported into the ear is left for actual storage in the grain.

2. The parasitogenically accelerated basal metabolism of the host. In loose smut of wheat (*Ustilago tritici*, p. 108) the infected grains 4 days after germination already produce about 20% more carbon dioxide, calculated on dry weight, than healthy grain. In the growing plant the respiration of individuals with latent infection, again calculated on the dry weight, remains consistently about 20% higher than that of healthy plants, and increases at the time of the paroxysm, i.e. with the appearance of the smutted ears, to about 40% (Kurssanow, 1928). Similar relations obtain in bunt of wheat. In loose smut, assimilation is also sometimes increased parasitogenically, but the general rule holds good that the additional carbohydrate is respired at the expense of the assimilatory metabolism instead of being stored in the endosperm.

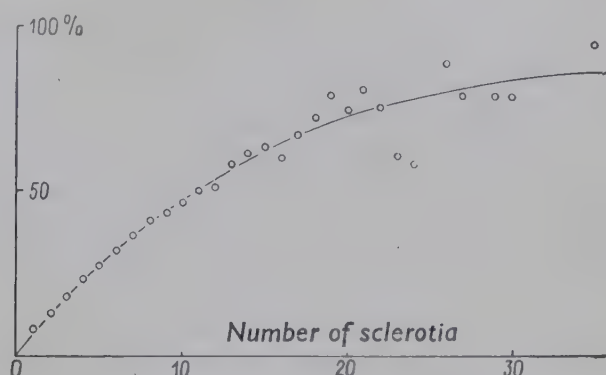


FIG. 296. The relation between the weight of ergots and the total grain weight; an example of the withdrawal of nourishment from a host by the parasite. Abscissae: number of ergots per ear. Ordinates: weight of ergots as a percentage of the total grain weight (rye grains plus ergot sclerotia) per ear. (After Krebs, 1936.)

Such an intensively respiring, i.e. intensively katabolic, individual cannot therefore produce so much dry weight, straw, and grain as healthy individuals with a balanced metabolism. The smaller yield (Fig. 297) is thus not only an expression of direct parasitogenic decrease in anabolism, of production of substance (e.g. assimilation), but also an expression of parasitogenic over-increase of basal metabolism. Certainly the equivalent of respiratory energy is work, but in this case it is uneconomic work. Sick men often lose weight in spite of abundant food because of their increased basal metabolism.

3. Interference with the anabolism of the host. Usually this is brought about by the direct destruction of a part of the assimilating leaf-surface (e.g. late blight of potatoes caused by *Phytophthora infestans*), or by functional disturbances of the production apparatus (e.g. by depression of assimilation, to be mentioned later, or by disturbance of growth substance economy), or by mechanical or functional disturbances in the transport of material (e.g. phloem necrosis, Fig. 281, foot rot of cereals, bast disease of trees).

The share taken by each of the three components in the lowering of productivity of the host has not yet been ascertained with sufficient

accuracy for any infectious disease of plants. In the same way it is still uncertain how the individual physiological component functions are modified by over-increase of basal metabolism and lowering of productivity through infection, and how the resulting changes influence one another. We will, therefore, consider the more important manifestations

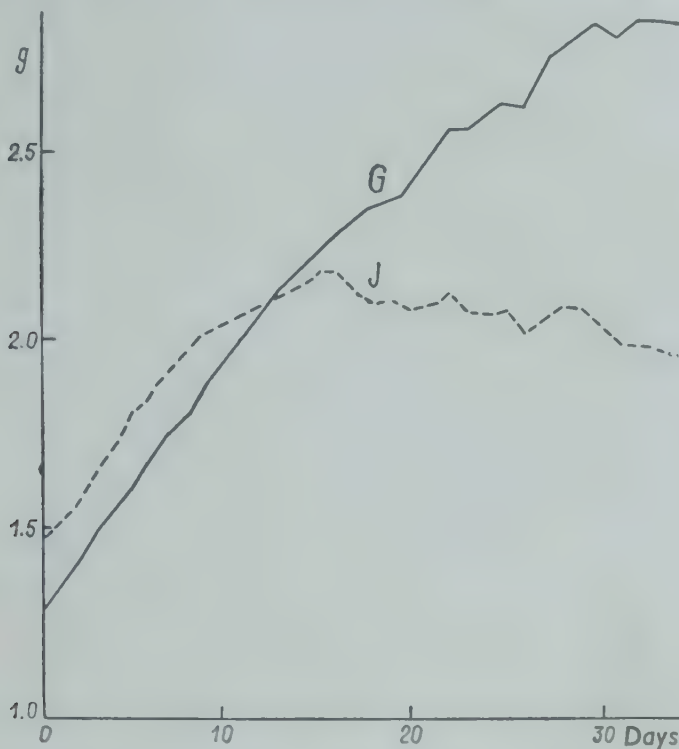


FIG. 297. The influence of brown rust infection (*Puccinia triticina*) on the growth-rate of two young wheat plants (var. Carina, which is susceptible to all the usual biotypes, as to type 3). Abscissae: days after infection. Ordinates: fresh weight of plant. Curve G: healthy plant. Curve J: infected plant. (After Werner and Steiner, 1933.)

separately: disturbances of (1) energy metabolism, (2) carbohydrate metabolism, (3) protein metabolism, (4) mineral metabolism, (5) water economy, (6) cell chemistry.

1. Disturbances of Energy Metabolism

The energy metabolism of an organism supplies first the maintenance energy (working energy, basal metabolism) and, in so far as excess energy is still available, the productive energy (growth and production of substance). In the course of pathogenesis (especially in morphoses) both the maintenance energy and the productive energy can be disorganized or misdirected, e.g. the smut boils of maize (Fig. 144). In the present connexion we are interested only in maintenance energy; its metabolism can be summarily determined on the basis of respiration (*Atmung*) and body temperature.

In the progressive phase of a disease there is usually an increase of energy metabolism, a heightening of respiration and tissue temperature; in the regressive phase there is usually a depression of both.

A simple example is afforded by potato tubers infected by *Bacillus atro-septicus* (black leg; Figs. 298, 299).

The normal body temperature of a healthy potato tuber is about 0.005°C . (i.e. about $5/1,000$ of a degree) warmer than the surrounding air. Naturally variety, previous cultivation, and time of year play their parts; the areas round the sprouts (the 'eyes') are somewhat warmer than



FIG. 298. Black leg of potato caused by *Bacillus atro-septicus*. (After Stapp, 1932.)

the intervening areas, and in spring the rose end is warmer than the stem end, &c. This excess average body temperature of 0.005°C . is shown in Fig. 299 as the horizontal line *KT*.

If the tuber is wounded mechanically an increased enzymic and growth activity ensues as a wound reaction in the affected tissues, and the temperature of the wound site rises within an hour to about fourteen times the normal body temperature (traumatic rise in temperature; Fig. 299, curve *WR*). Then healing and cork formation begin, the wound temperature at first falls rapidly, then more slowly, and finally returns to normal after about 2 days: the wound is healed.

If the wound site is infected by *Bacillus atro-septicus* the healing process is interrupted and is replaced by an infectious disease which spreads farther and farther in the neighbouring tissues. This disease leads to a local increase of metabolism. The diseased tissues produce up to nine times as much carbon dioxide

per unit of time and area as do the healthy tissues (parasitogenic increase of respiration; Fig. 299, curve CO_2). By this increased katabolism more heat is set free than can be given out immediately. Consequently the temperature of the wounded and now infected tissue does not sink any farther but again begins to rise (direct parasitogenic increase in temperature; Fig. 299, curve *Inf.*), and after about 170 hours, i.e. about a week, reaches its highest point, which is about thirty to thirty-five times the normal body temperature. Then, in the case of the example in Fig. 299, the infected tissues begin to die and the tissue temperature and respiration gradually sink to zero.

This raising of metabolism is not limited to the tissues immediately infected, but also involves the healthy tissues of the tuber. Thus, in Fig. 299,

the increase of temperature in the non-infected tissue 2.2 cm. from the infection site (curve 2.2 cm.) is about sixteen times the normal body temperature, and at a distance of 4.4 cm. from the infection site is still about eight times as high. This indirect parasitogenic rise of temperature may be called tissue fever, since fever is the pathological raising of the temperature of tissue not itself diseased.

Fig. 299 shows two important points. First, that the whole tuber undergoes a heightening of metabolism due to a local infection, and, accordingly, the tuber as a whole goes through a disease process. Since this increase

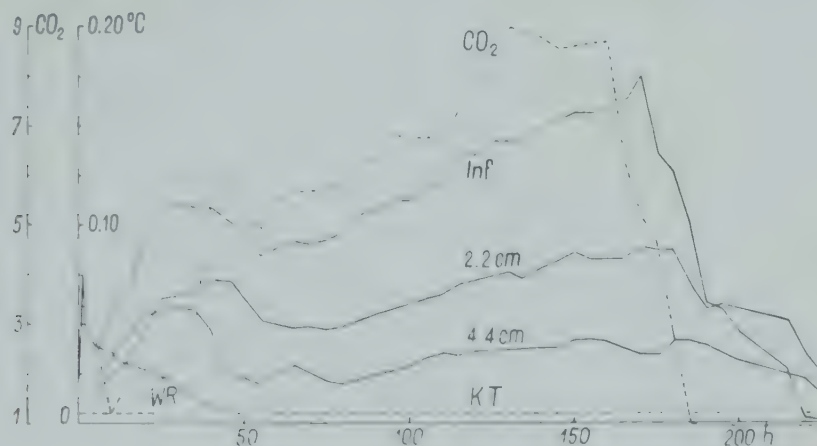


FIG. 299. The variation in tissue temperature and respiration-rate in a potato tuber, var. Paulsens Juli, during infection by *Bacillus atrosepticus*. *KT*: normal temperature of intact tuber. *WR*: traumatic rise in temperature (temperature curve of wound reaction). *Inf.*: direct parasitogenic rise in temperature (temperature curve in the immediately infected tissue). 2.2 cm. and 4.4 cm.: indirectly parasitogenic rise in temperature (temperature curve of healthy tissue 2.2 cm. and 4.4 cm. from infection focus). *CO₂*: parasitogenic increase in respiration. The proportionate figure shows how much more *CO₂* per unit surface area is given off from diseased than from healthy tissue. (After Eglits; from Gäumann, 1933.)

in metabolism only becomes evident after the ability of the cell to maintain equilibrium has been overtaxed, a whole chain of cellular pathological events must take place in the healthy tuber tissues before the parasitogenic disturbance of its metabolism becomes pronounced enough to be measurable. Some of these cellular pathological processes in the healthy tissues are presumably effective against the pathogen or its toxins and, therefore, are of the nature of anti-infectional or antitoxic defence reactions.

The second important feature in Fig. 299 is that the intensity of the response decreases with increasing distance from the local focus of infection, evidently because the intensity of the stimulus also diminishes with increasing distance. In contrast to ordinary 'fever' of the human body, in the potato tuber the rise in temperature, the hyperthermy, is not the same in all tissues, but evidently depends upon the strength of the stimulus to which they are immediately subjected. This divergent behaviour in plant and man depends upon their different forms of organization. The human body has two types of pathological increase in temperature, a local and a general.

A local or tissue hyperthermy (calor) arises, for example, in carbuncles, in the immediate neighbourhood of the local focus, and decreases rapidly with distance. Medically this is unimportant, but it has biological significance because it is homologous with the tissue increases in temperature in infectious diseases of plants.

In general or centrally controlled hyperthermy the toxins of the parasite or the body's own decomposition products stimulate the heat regulating centres in the mid-brain and fore-brain, and thereby lead indirectly to that uniform, thermal, systemic reaction of the whole organism which we term 'fever'. 'Fever' of the human body accompanies tissue hyperthermy of

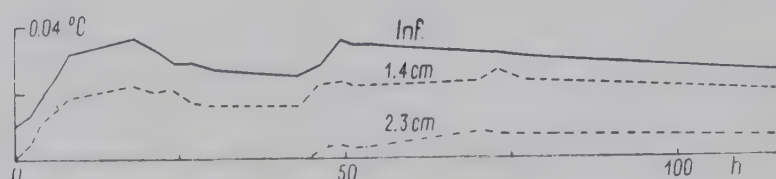


FIG. 300. The tissue hyperthermy in a potato tuber, var. Bintje, after infection by *Phytophthora infestans* (late blight). *Inf.*: direct parasitogenic rise in temperature at the site of infection. 1.4 and 2.3 cm.: indirect parasitogenic rise in temperature in tuber tissue 1.4 and 2.3 cm. from the focus of infection. (Orig. Gäumann and Fischer.)

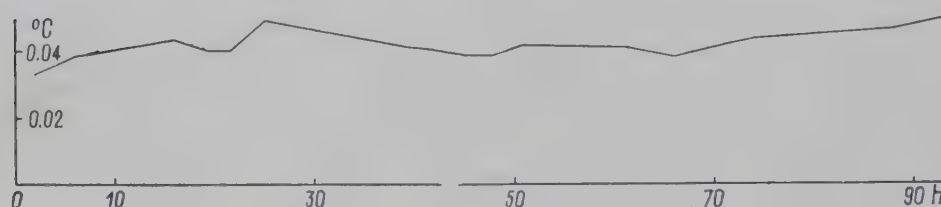


FIG. 301. The tissue hyperthermy in a potato tuber, var. Bintje, at the site of infection with a *Fusarium* similar to *F. solani*. (Orig. Gäumann and Fischer.)

the infection focus, is superimposed upon this and, since it depends upon a parasitogenic excitation of certain parts of the central nervous system, it must be absent from the plant kingdom.

Similar tissue hyperthermy is caused by infection, for instance, of potato tubers by *Phytophthora infestans* (late blight; Fig. 300) and *Fusarium solani* (white rot; Fig. 301). Because of the milder course of these infections the temperature effects are less marked than in the case of *Bacillus atrosepticus*.

In the same way, parasitogenic increases in respiration, as shown in Fig. 299, have been measured in numerous mycoses and viroses, e.g. of plants and leaves. In these diseases there is, in general, an increase in respiration, i.e. in energy metabolism, only between about 20 % and 100% (e.g. Fig. 302), but in mildewed leaves of wheat it can reach 650% (Allen and Goddard, 1938). It is thus, as a rule, less in the potato tuber, which is scarcely surprising because in the latter, which is a physiologically inert storage organ, the normal respiration which serves as the basis of comparison is very low.

That this increased respiration depends on stimulation of the host and

not on the respiration of the pathogen itself is shown, for instance, by measurements on mildewed plants. The Erysiphaceae are ectoparasites which usually send their haustoria only into the epidermal cells (Fig. 76) and, therefore, can be removed with a fine brush or destroyed by chemical means.

In young wheat plants attacked by *Erysiphe graminis tritici* the oxygen consumption is 7.9 c.mm. per sq. cm. of leaf surface per hour compared with only 1.7 c.mm. in healthy leaves; the pathological increase in oxygen consumption thus amounts to 6.2 c.mm. If the fungus mycelium is removed mechanically from the diseased leaves the oxygen consumption

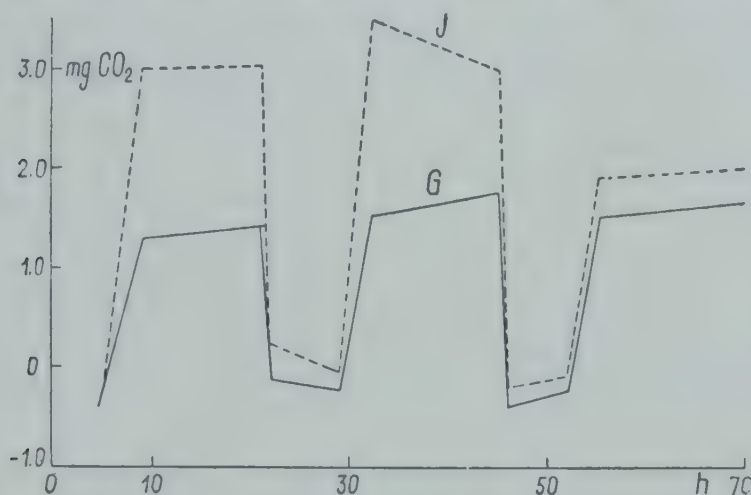


FIG. 302. The respiration of healthy and leaf roll infected potato plants with alternation of light (curve minima) and dark (curve maxima). Abscissae: duration of the experiment in hours. Ordinates: mg. of CO_2 output per hour per g. dry weight. Curve G: healthy plant. Curve J: plant infected with virus. (After Whitehead, 1934.)

falls only from 7.9 to 6.4 c.mm. per sq. cm. of leaf surface. From this two conclusions may be drawn: (1) Of the total pathological increase in oxygen consumption (6.2 c.mm.), at most 1.5 c.mm. can be ascribed to the respiration of the fungus mycelium, the remaining 4.7 c.mm. are derived from the host. (2) The heightened respiration of the leaf tissues persists even when the pathogen has been mechanically removed; the parasitogenically induced state of excitation of the host continues as an after-effect.

Evidence for the existence of a special respiration-increasing effect emanating from the pathogen is also afforded by the way in which the pathological oxygen consumption is distributed in the leaf tissue. The directly infected epidermis uses only 0.14 c.mm. of oxygen; hence, the main part of the parasitogenic increase of metabolism is borne by the uninfected, only indirectly stimulated mesophyll (Allen and Goddard, 1938).

Observations on white clover (*Trifolium pratense*) infected with powdery mildew (*Erysiphe Martii*) point in the same direction. In the course of 9-11 days the respiration of the clover leaves increased by about 40% (in infection with clover rust, *Uromyces fallens*, even by 123%) and remained

at this level after the mycelium of the mildew had been killed by chemical means (Yarwood, 1934). Thus, in this case also, the increased respiration is induced in the host by chemical stimulation from the pathogen.

The chemical nature of the substance which activates respiration varies. In *Gibberella Saubinetii* (brown foot rot and ear blight of wheat) the substance is stable, water soluble, and possibly closely related to pantothenic acid (Hellings, 1941); it is non-specific in origin and is also formed,

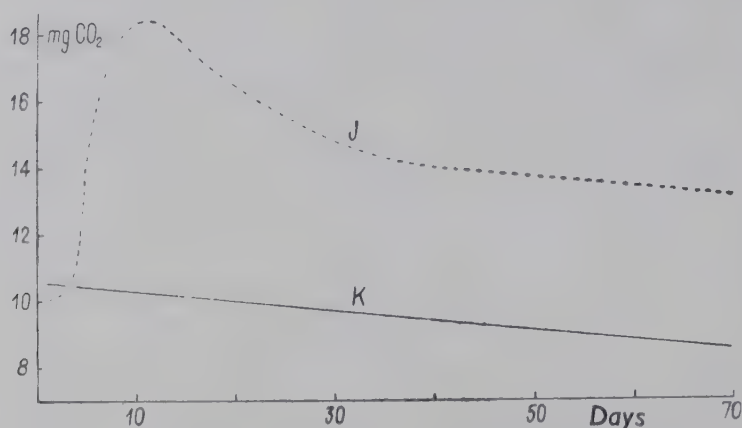


FIG. 303. The stimulatory effect of a lemon infected with *Penicillium digitatum* on the respiration of 50 healthy lemons. Abscissae: days after the start of experiment. Ordinates: mg. of CO_2 given out per kg. of fruit per hour. Curve J: CO_2 output in the presence of the gaseous emanation. Curve K: control; CO_2 output in a parallel series without the pathogen. (After Biale and Shepherd, 1941.)

for example, by saprophytic fungi like *Trichoderma lignorum* and by yeasts: it is also non-specific in its effect since, for example, it also stimulates respiration in potato tissues. On the other hand, *Penicillium digitatum* (green mould of citrus fruits) excretes a gaseous, respiration-stimulating substance (Fig. 303) so that the presence of a few infected fruits in a store room is enough to bring about a considerable amount of damage to all the other fruits. This course of events is also noticeable in the winter storage of apples.

2. Disturbances of Carbohydrate Metabolism

Parasitogenic disturbances in carbohydrate metabolism manifest themselves in an altered rate of assimilation or in an abnormal distribution of carbohydrates and fats in the tissues of the host.

In one and the same host assimilation can be furthered by one parasite and hindered by another; thus, in wheat, it is raised by 20–30% by the mycelium of loose smut (*Ustilago tritici*) (Kurssanow, 1928), whereas, on occasion, it is reduced to one-third by rust attack (Fig. 304). It is characteristic of rust attacks that during the incubation period (about 8 days) there is no serious disturbance of assimilation; the downward bend of the curve in Fig. 304 coincides with the manifestation of injury in the host tissues. Its decline continues during the sporulation of the pathogen which begins after 10–12 days.

Because of the small number of examples which have been investigated no general conclusions regarding parasitogenic changes of assimilation are yet possible.

The abnormal distribution of carbohydrates in the host tissues shows itself either (1) in a quantitative change, or (2) in a qualitative conversion into another form, or (3) in a local accumulation.

1. Quantitative changes in carbohydrate content can follow a positive

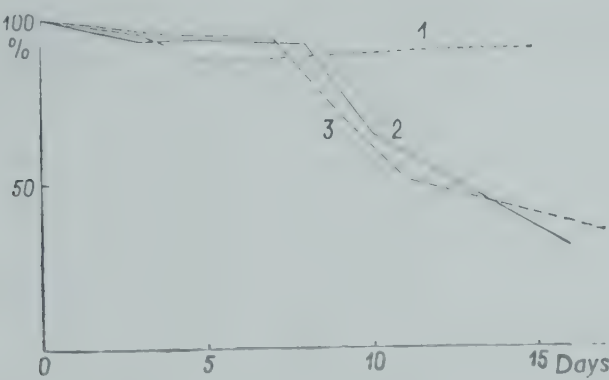


FIG. 304. The influence of yellow rust attack on the assimilation-rate of wheat. Abscissae: days after infection. Ordinates: assimilation-rate of infected leaves expressed as a percentage of that of healthy leaves. Curve 1: highly resistant variety (reaction type i). Curve 2: variety with moderate susceptibility (reaction type O-II). Curve 3: highly susceptible variety (reaction type IV).
(After Gassner and Goeze, 1936.)

or a negative trend (e.g. Fig. 305). As a rule the pathogen’s own consumption and the increased respiration of the host tissues lead to a quantitatively measurable decrease, the amount varying with the severity of attack (Table LXXXVI).

TABLE LXXXVI

The influence of maize smut (*Ustilago zae*) on the carbohydrate content of infected internodes. (After Hurd and Hasselbring, 1927)

Incidence of attack	Dry matter		Reducing sugar as glucose		Sucrose		Total sugar as glucose	
	healthy	diseased	healthy	diseased	healthy	diseased	healthy	diseased
	%	%	%	%	%	%	%	%
Severe attack	17.1	14.5	11.5	2.1	22.8	3.7	35.5	6.0
Moderate attack	17.5	17.5	9.5	8.3	7.6	6.6	17.5	15.2
Slight attack	22.8	22.8	14.1	12.1	13.8	9.9	28.6	22.5

In cultivated plants, which are grown just because of their carbohydrate content (e.g. potatoes and sugar beet), economic damage can arise in this way. In other cases, however, the loss of sugar can result in a certain commercial advantage; thus, in grey mould of grapes, caused by *Botrytis cinerea*, there is a marked decrease in the sugar; for instance, in a particular experiment 11.7% of the original sugar had disappeared from the berries

by 15 October and 19.6% by 30 November (Müller-Thurgau, 1888). But, since the water content of the berries falls even more than their sugar content, the late vintage must from grey moulded grapes is richer in sugar and thus more valuable than that from ordinary fully ripe grapes.

2. Qualitative change in carbohydrates (conversion into another form) is induced either directly by the pathogen or indirectly by a change in the metabolism of the host.

Enzymatic changes initiated by the pathogen arise in connexion with the previously mentioned withdrawal of nutrient material. The pathogen secretes enzymes which break down the polymerized carbohydrates and transform them into a more easily assimilable form. Naturally the dosage

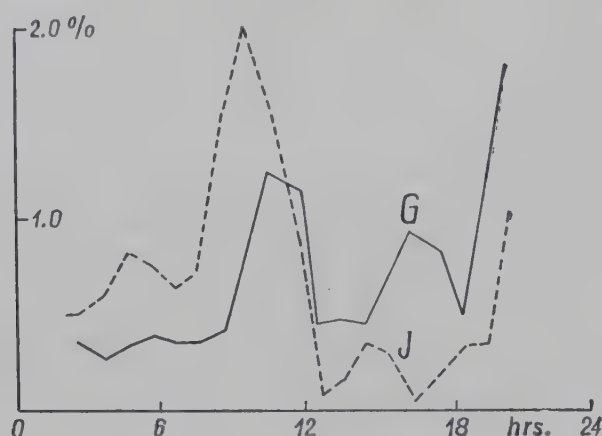


FIG. 305. The influence of a virosis (Crinkle, p. 227) on the carbohydrate content of potato plants (var. Arran Victory). Abscissae: time of day. Ordinates: average saccharose content of the leaf stalk. Curve G: healthy plants. Curve J: diseased plants. (After Barton-Wright and McBain, 1933.)

of these enzymes is not such that they only work on just that amount of material which is immediately required; usually, since otherwise the pathogen would starve, there occurs an over-production of enzymes and correspondingly a surplus of the split products. In tissues attacked by fungi there sometimes results a temporary increase in the invert sugar content at the expense of the sucrose, or of the invert sugar content and sucrose content at the expense of the starch.

A temporary increase in the invert sugar content at the expense of the sucrose is found in sugar beet and certain fruits. In sugar beet, species of *Botrytis* split off an invert sugar which technically is of little value, so that an economically valuable kind of sugar, sucrose, has been converted by the parasite into one of economically lower value. In peaches, *Sclerotinia cinerea* (brown rot) inverts the sucrose to the point of almost complete disappearance: in one instance it diminished from 2.51 to 0.16% but, at the same time, the content of the tasteless reduced sugar (reckoned as glucose) increased from 1.65 to 3.82% (Hawkins, 1915). Thus, the inverted sugars are, at first, used by the parasite only to a small extent for its own needs; they remain available but in another form.

A temporary increase in the total sugar content of the tissues at the

expense of the starch is initiated in potato tubers by *Phytophthora infestans*; here the sucrose content increased in a particular example from 0.54 to 2.55% and the invert sugar from 0.4 to 0.65% (Lepik, 1929).

Similar excess splitting sometimes occurs in the biological decomposition of wood; certain rot fungi decompose more wall-substance than they actually need at the time and so help to nourish secondary micro-organisms which follow them.

The second form of qualitative change of carbohydrate by means of an alteration in the metabolism of the host is, for example, produced by *Uromyces betae* (beet rust) in the leaves of sugar beet: instead of starch, fat is produced in the attacked tissues and, indeed, in such amounts that the fungus can only be recognized microscopically after special pre-treatment (Schmidt, 1932).

3. A local accumulation of carbohydrates occurs, particularly in localized infections, in the zone of conflict around the focus of infection (e.g. in fungal galls, &c.) and sometimes in such quantity that they can be determined not only microscopically or micro-chemically but by direct weighing. In sunflowers attacked by rust (*Puccinia helianthi*), the dry weight of the whole plant in a progressive attack of the disease falls even farther behind that of the non-infected controls, 11 days after infection averaging 3.4%, 20 days after infection 13.9%, and 28 days after infection 41.1% (Yarwood and Childs, 1938). But, in some rust diseases, if one compares corresponding portions of tissue from paired leaves of which only one has been infected, the dry weight per sq. cm. in the obviously diseased zone proves to be higher than in the controls (Table LXXXVII).

TABLE LXXXVII

The influence of rust attack on the dry weight of portions of leaf tissue.
(After Yarwood and Childs, 1938)

Parasite	Host plant	Dry weight of leaf tissue per sq. cm.		Increase
		healthy leaf	diseased leaf	
		(mg.)	(mg.)	%
<i>Tranzschelia pruni spinosae</i>	<i>Prunus domestica</i>	840	853	1.5
<i>Uromyces appendiculatus</i>	<i>Phaseolus vulgaris</i>	815	968	18.8
<i>Puccinia helianthi</i>	<i>Helianthus annuus</i>	227	273	20.5
<i>Uromyces betae</i>	<i>Beta vulgaris</i>	955	1,219	27.7
<i>Puccinia antirrhini</i>	<i>Antirrhinum majus</i>	1,346	1,809	34.4

In general, the same substances are laid down in the zone of conflict as the host deposits in its own storage organs; sugar in peach leaves with leaf curl (*Taphrina deformans*) and in pear leaves with blister disease (*Taphrina bullata*); inulin in leaves of *Tussilago Farfara* after attack by *Puccinia poarum*; and mucilage and dextrin in leaves of *Sempervivum* spp. after attack by the house leek rust (*Endophyllum sempervivi*) (Schellenberg, 1911).

These carbohydrates are not due to a parasitogenic increase of assimilation but are withdrawn from the sound tissues. Within its sphere of influence, the pathogen changes the selective permeability of the protoplasmic membranes and the osmotic, &c., qualities of the cells and thereby creates the conditions which ensure the supply of the products of assimilation to the focus of infection. For instance, pea rust (*Uromyces fabae*) increases the permeability of pea leaves to sucrose (Thatcher, 1939).

At the focus of infection the assimilates first provide for the increased energy economy of the host and the pathogen, since a heightened sugar concentration is necessary for the continued parasitogenic increase of respiration, discussed on page 465. But they also provide the material basis for the cellular defence reactions of the host tissue; later, however, they constitute the material basis for the sporulation of the pathogen, thus subserving a purpose not their own, and they are first made use of, for example, by the parasitic fungi during the formation of their reproductive bodies. Nevertheless, the draining of these parasitogenic storage tissues is never as complete as in the normal storage organs, and when the local focus of infection dies, the material not used by the fungus does not return to the healthy parts of the host. Thus, a wastage of assimilates occurs due to the pathogen.

Finally, certain viroses constitute a special cause of local accumulation of carbohydrates, as in leaf roll disease of potatoes. Because of enzymatic disturbances and difficulties in transport consequent on degeneration of the phloem, the products of assimilation are stored in more solid form (starch) directly in the assimilating leaf tissue, so that the foliage leaves are packed full of starch grains in spite of increased respiration (Fig. 302) and reduced photosynthesis.

3. Disturbances of Protein Metabolism

The nitrogen economy of diseased tissues and individuals conforms to the same rules as the disturbance of carbohydrate metabolism just discussed. The disease may be accompanied by a quantitative decrease or by a qualitative change in the nitrogen-containing components to another form, or by an increase in nitrogenous compounds. In consequence, the nitrogen/carbohydrate ratio in the diseased tissues and individuals may vary in divers ways.

A quantitative decrease in the host's own (native) nitrogen compounds is found especially in the viroses. Either the viruses multiply chiefly at the expense of the protein compounds of the host, or the virus protein is mostly formed by the host itself. In the latter case, the diseased state termed a virosis consists in the organism allowing itself to be forced into aberrant protein syntheses. The total protein content of tobacco plants suffering from mosaic may, for instance, be about twice that of normal plants, of which the virus protein may amount to 80% (Woods and Du Buy, 1941).

To a smaller extent pathogenic fungi and bacteria also withdraw from

the host tissues nitrogenous compounds which they use in the building of their own body substance. But the two disparate moieties, that belonging to the host itself and that withdrawn from the host by the pathogen and built into its own body, can be differentiated by mass analyses only where their origin can be inferred from other characteristics. For example, *Phytophthora infestans* breaks down the amino-acids, &c., of attacked potato tubers and stores their equivalent in its own mycelium (p. 387).

A qualitative change of nitrogenous compounds occurs in celery leaves attacked by *Cercospora apii* and by *Septoria apii*. The former causes mainly a spotting of the leaves which may occupy up to a sixth of the leaf area, and the latter frequently kills the leaves, or the greater part of them, giving rise to a characteristic discoloration (celery leaf spot). In both diseases the affected plants have a lower nitrogen content than the healthy ones; in sampled material attacked by *Cercospora* the amount of nitrogen was 61% of that in healthy material, and attacked by *Septoria*, 83%.

Different nitrogen compounds are not affected by these two pathogens in the same way (Coons and Klotz, 1925), so that the disturbance of the nitrogen balance is, to some extent, characteristic for each pathogen. In *Cercospora* attack the fall in nitrate nitrogen, relative to the total nitrogen content, amounts to 3.4%, but in *Septoria* attack only to 0.1%. In *Cercospora* attack the decrease in total nitrogen hydrolysable by hydrochloric acid is 2.6%, and in *Septoria* attack 3.6%; on the other hand, ammonium nitrogen and protein nitrogen undergo an increase relative to total nitrogen by 1.3 and 6.1% respectively in the case of *Cercospora* attack, and 1.0 and 4.8% respectively in the case of *Septoria* attack. The significance of these changes is still obscure.

TABLE LXXXVIII

Changes in the nitrogen metabolism of tomato stems caused by bacterial canker. (After Klein and Keyssner, 1932)

Nitrogen compound	100 g. dry material contain mg.		
	above the tumour	in the tumour	below the tumour
Protein-N	475	1,290	422
Soluble N	185	711	268
Ammonium-N	11	18	6
Amide-N	20	46	33
Amino-N	88	228	85

A considerable local increase in nitrogenous compounds has frequently been observed in bacterial plant galls, where the tumour tissues often contain three times as much dry weight nitrogen as the adjoining healthy tissues (Table LXXXVIII). A comparable selective accumulation of nitrogen to the detriment of the host occurs in mistletoe (Table LXXXIX) which, in France, is sometimes used as cattle food because of its high protein content. In addition to the technical devaluation of the trunk due

to the mistletoe sinker (the timber can be used neither for veneering nor boarding) there is the damage which the mistletoe does to the host by this removal of enormous amounts of nitrogen.

This withdrawal of nitrogen from healthy tissues is facilitated by the circumstance that certain pathogens, within their sphere of influence, appreciably raise the selective permeability of plasma membranes to nitrogen compounds as they do to carbohydrates (p. 474); thus carnation

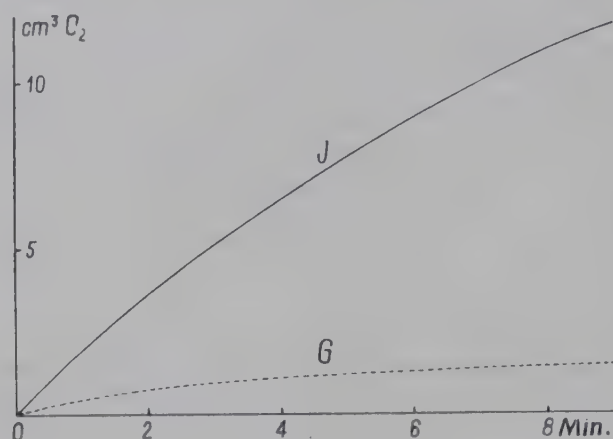


FIG. 306. Increase of permeability to urea in carnations after infection by *Uromyces caryophyllinus* (carnation rust). Abscissae: time in hours for deplasmolysis to occur in a 1.5 molar solution. Ordinates: percentage deplasmolysis. Curve *G*: non-infected tissue. Curve *J*: infected tissue. (After Thatcher, 1939.)

rust (*Uromyces caryophyllinus*) increases the permeability to urea in carnations (Fig. 306).

4. Disturbances of Mineral Metabolism

In the same way that the pathogen in some cases modifies the selective permeability of plasma membranes to carbohydrates, so it sometimes alters it to certain mineral salts, thereby bringing about an accumulation or depletion of particular ash constituents in the diseased tissues. This field has, as yet, been little explored. Perhaps these changes are also specific; thus, *Bacterium tumefaciens* tumours on potatoes seem to have a higher ash content than healthy tissues (in particular of potassium and phosphorus with unaltered calcium), while *Synchytrium* tumours have a lower ash content (especially of potassium and phosphorus, with a slightly raised calcium content).

Naturally, part of the ash constituents of the tumour must have been taken up by the pathogen itself and stored in its body, i.e. withdrawn from the mineral economy of the host, but it is scarcely possible to estimate this portion analytically. Only in the case of external phanerogamic parasites is it possible to show that their haustoria have a capacity of selective absorption, since, for instance, their ash is richer in potassium and phosphorus and poorer in calcium than that of their hosts (Table LXXXIX).

5. *Disturbances of Water Economy*

The water relations in diseased tissues or individuals may be influenced directly or indirectly by disease.

Direct disturbances of water economy usually manifest themselves in increased transpiration. This comes about either physically, or physiologically in connexion with the general acceleration of metabolism in the diseased tissues.

TABLE LXXXIX

Ash contents of phanerogamic parasites compared with those of their substrate. Nitrogen and ash contents are relative to dry weight, the ash fractions relative to total ash content. (After Nicoloff, 1923)

Portion analysed	Nitro- gen %	Ash %	P ₂ O ₅ %	K ₂ O %	CaO %	MgO %
<i>Mistletoe (Viscum album) on Pirus Malus</i>						
Mistletoe	26.4	6.1	14.5	36.0	10.9	5.2
Branch immediately below the parasite	3.5	1.9	7.5	11.1	43.9	5.3
Portion of branch distal to parasite .	3.5	2.8	3.7	8.4	78.6	3.8
Portion of branch proximal to parasite	4.7	4.4	3.1	6.7	52.1	2.9
<i>Orobancha ramosa on Nicotiana tabacum</i>						
Orobancha	15.8	9.3	6.5	37.2	1.4	2.0
Roots of parasitized tobacco plants .	21.6	16.3	2.2	18.2	20.4	5.1
Roots of non-parasitized tobacco plants	21.8	16.7	3.2	22.7	14.3	4.2
<i>Cuscuta europaea on Sambucus Ebulus</i>						
Cuscuta	20.0	5.1	15.4	51.7	1.3	4.1
Parasitized <i>Sambucus</i> leaves . . .	21.4	9.7	5.0	15.6	29.2	11.5

The conditions for a purely physical increase of water loss are created, for example, by the destruction of the outer layers which reduce evaporation, e.g. the cuticle and epidermis of fruits and the periderm of potatoes. As the water supply does not suffice to replace completely the amount of water transpired away, the water content of the tissues in time diminishes (loss of weight in fruits!) and the dry matter content accordingly rises. Finally the diseased tissues shrink, and this may take place quite early on the parent plant in stone and berried fruits attacked by *Monilia* and in grey moulded grapes (*Botrytis cinerea*), whereas, in stored fruit (apples) and potatoes, it occurs only during winter storage.

A physiological increase of transpiration connected with accelerated metabolism probably occurs in the progressive phase of all infectious diseases of plants. Thus all infected tissues, and those immediately adjoining the infection focus, transpire more than normal tissues (Fig. 307). The extent of this rise in transpiration will depend, *ceteris paribus*, on susceptibility, i.e. on severity of attack.

The increased rate of water loss, however, can only be sustained if the ability of the conducting tissues to supply water is also increased by the parasite. This effect has been demonstrated in leaves of the pea (*Pisum*

sativum) after infection by rust (*Uromyces fabae*), and in celery roots after infection by *Botrytis cinerea* and *Sclerotinia sclerotiorum* (Thatcher, 1939). As the last two fungi are highly toxigenic, they increase the permeability of the celery tissues to water up to a distance of about 12 cm. (Table XC).

TABLE XC

Increase in permeability to water of celery tissues (Apium graveolens) after infection with Botrytis cinerea and after infection with Sclerotinia sclerotiorum, measured by the average time for deplasmolysis after having been plasmolysed in calcium chloride. (After Thatcher, 1939)

<i>Distance from necrotic zone</i>	<i>Local infection with Botrytis cinerea</i>	<i>Local infection with Sclerotinia sclerotiorum</i>
(mm.)	(sec.)	(sec.)
7.5	94	118
30	149	139
60	—	148
120	323	390
Petiole not infected	477	

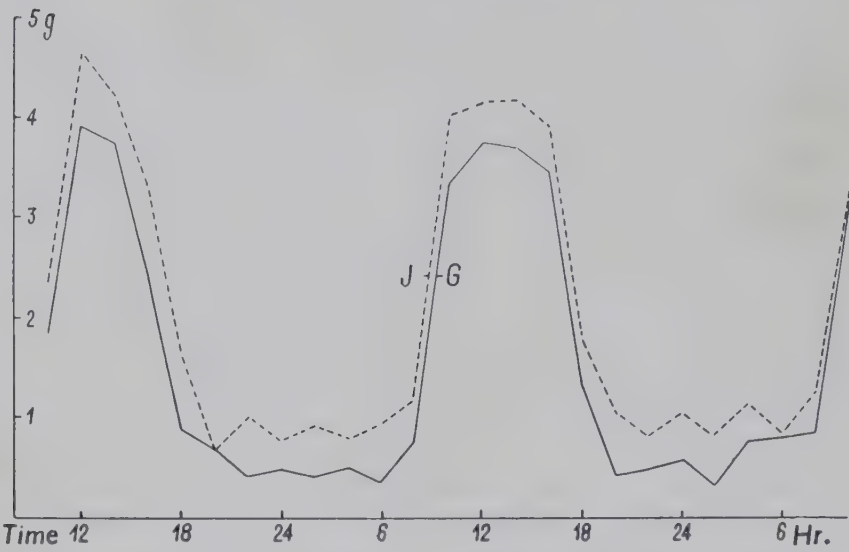


FIG. 307. The influence of brown rust attack (*Puccinia triticina*) on the daily course of transpiration in mature plants of the highly susceptible wheat var. Pusa No. 4. Curve G: healthy plants. Curve J: diseased plants. Abscissae: time of day during 48 hrs. Ordinates: average water-loss per plant and hour. (After Johnston and Miller, 1940.)

In contrast to direct disturbances in water economy, the indirect disturbances do not usually manifest themselves primarily in excessive water loss but in a diminution of water supply, so that a water deficit occurs in the foliage in spite of adequate water reserves in the soil.

The water supply may be cut off purely mechanically. For instance, *Fusarium Martii phaseoli* (wilt of beans) destroys the root neck and the roots of infected individuals, thereby inevitably causing a water deficit on warm days. In the *Endothia* disease of chestnuts the restriction of the

water supply is not quite so crudely mechanical but is an indirect histological effect. The exudates of the pathogen stimulate the host to produce abundant tyloses (Fig. 308) which block the vessels, so that the crown begins to suffer from lack of water.

In the typical wilt diseases the disturbance of water economy is not effected mechanically, but the whole physiological system of the host is paralysed by toxins (p. 242). The pathogenic organisms (mostly species of *Fusarium* and *Verticillium*) penetrate the roots or stem base, grow into

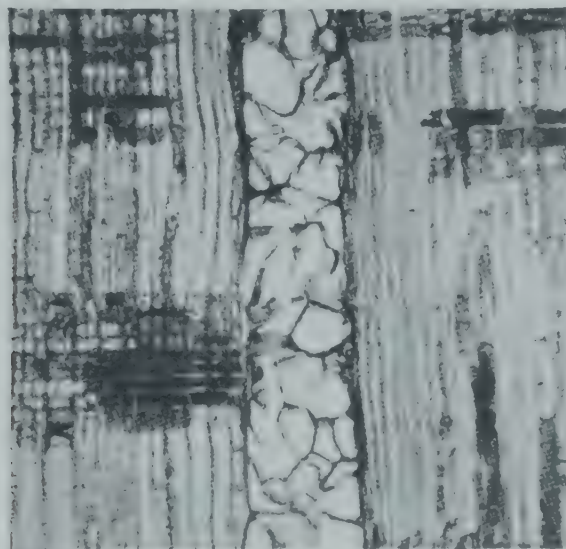


FIG. 308. Blocked vessel from the youngest annual ring of a chestnut tree attacked by *Endothia parasitica*, showing tyloses whose formation was induced by the parasite. The medullary rays have been directly infected; their tannins have been oxidized. $\times 94$. (After Bramble, 1938.)

the vessels (tracheomycoses), inducing in them a series of histological reactions (Fig. 309) which, themselves, tend to make the water supply difficult.

6. Disturbances of Cell Chemistry

In addition to the macrochemical effects already considered, every pathogen initiates numerous metabolic physiological changes which manifest themselves only in individual cells. Some of these changes, e.g. in growth substance economy, in enzymatic activity, in acidity of the cell sap and suction pressure, constitute the immediate pre-condition for the functional disturbances so far discussed, but others, e.g. pathological pigment formation and excretion of resins and gums, are the result of these and are sometimes the first indication of functional disturbance.

An increase of growth substance content occurs, for example, in bacterial crown gall of plants (p. 455), a decrease in some viroses (Fig. 310); thus, the growth substance content of degenerate (mostly X-virus infected) potato plants and tubers may fall by a half (Söding, 1942).

Changes in enzymatic activity have been followed mainly in bacterial tumours. Relative to the fresh weight, the catalase activity of sap extracted

from tumours on tomato stems is about 160% higher than in corresponding material from healthy stems, the oxidase activity is about 130% higher, and the peroxidase activity about 120% higher. Furthermore, the tumour tissues contain abundant tyrosinase, which is lacking in healthy tissues (Nagy *et al.*, 1938). The divergence in the catalase activity of beet root tumours is even greater (Fig. 311).

It may be shown by appropriate experimentation that increased catalase activity, for instance, is a specific phenomenon of tumour tissue, not merely

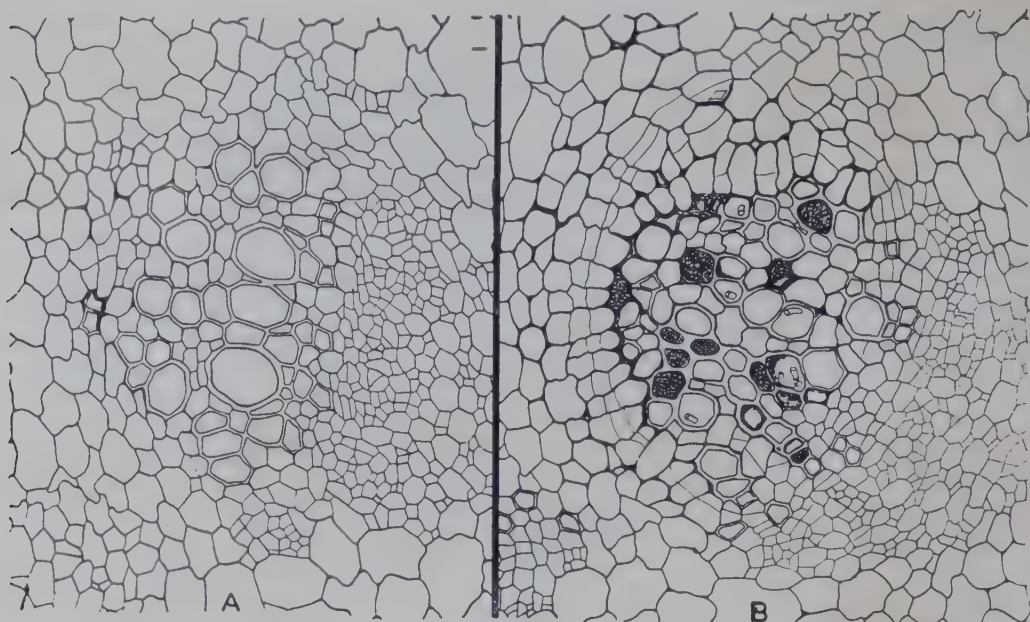


FIG. 309. Histological effects of wilt disease of pea (*Pisum sativum*) due to *Fusarium orthoceras* var. *pisi*. *A*, section through a healthy, *B*, through an infected vascular bundle. Gummy, granular, and deeply staining substances have been deposited in the tracheids. Cell walls, especially near the edge of the bundle, stain deeply; the xylem has become surrounded secondarily by a periderm-like layer. $\times 203$. (After Linford, 1931.)

of wound tissues, and that it is due to increased enzyme production by the host (Klein and Ziese, 1932).

If the reaction of the cell sap of diseased tissues is altered by the parasite, it usually becomes more acid by about 0.2–0.3 pH units; this represents a substantial change in the substrate for all physiological activity. In some cases, e.g. bacterial tumours, the hydrogen-ion concentration is not measurably affected (Nagy *et al.*, 1938). However, owing to the serious methodological difficulties, the results of investigations in this field are often contradictory.

The necessity for the suction pressure of the pathogen to be higher than that of the host has already been discussed (p. 271). In addition, the pathogen causes changes in the suction pressure of the affected tissues themselves, which may result in varying degrees of deflexion of the course of the annual periodicity.

The peach leaf curl disease (*Taphrina deformans*) causes a rise in suction pressure in peach leaves. In the spring the difference between healthy and infected leaves is still insignificant (Heusser, 1917) but, at about the

time when the leaves begin to curl, the osmotic value for diseased leaves begins to rise considerably, as measured by the limit of plasmolysis, and finally reaches about 5.5 atmospheres. During sporulation the healthy and diseased tissues again begin to resemble one another because the osmotic pressure of the healthy leaves increases steadily during their development, whereas that of the diseased tissues falls, so that the two curves eventually approximate.

On the other hand, a decrease of suction pressure from 9–11 to 6–8 atmospheres is caused by pea rust (*Uromyces fabae*) in the leaves of *Pisum sativum* (Thatcher, 1939).

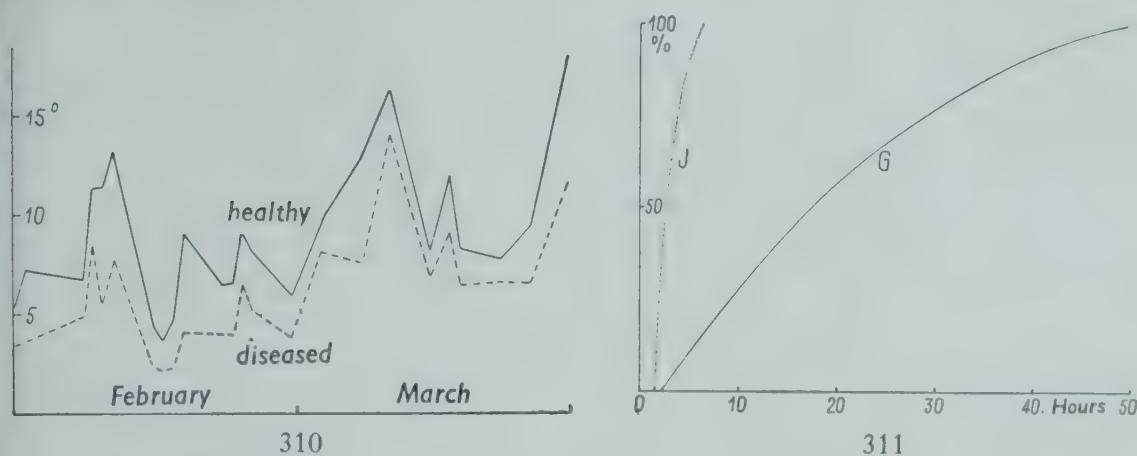


FIG. 310. Growth substance contents of healthy and virus-diseased potato tubers during early spring. Ordinates: angle of curvature in the *Avena* coleoptile test. (After Söding and Funke, 1941.)

FIG. 311. Catalase activity of tissue mash of beet root (curve *G*) and of its bacterial tumour (curve *J*). (After Klein and Ziese, 1932.)

Occasionally, the end result of aberrant reaction chains in the affected tissues or in their neighbourhood may become visible externally as pathological pigment formation whose mere occurrence implies the presence of disease (e.g. *Rotbrennen* of vines, p. 47). In plant pathological literature the red and violet pigments thus produced are summarily termed anthocyanins but, where they have been analysed chemically, as in oak galls (Nierenstein, 1919) and *Eriophyes* spots on *Eucalyptus stricta* (Petrie, 1924), no trace of anthocyanins has been found. Hence, the red pigments of diseased plants are not in every case identical with anthocyanins.

Finally, abnormal resin or gum formation can occur in disease as it does after injuries. The exudation of resin in conifers following attack by the honey agaric (*Armillaria mellea*) is often so copious that in Holland it is called *totbluten* (bleeding to death). Spruces attacked by *Trametes radiciperda* (annosus butt rot) and white pines attacked by *Cronartium ribicola* (blister rust), &c., react with similar pathological exudations of resin. Contrary to the view often held, these secretions have no defensive character, since the pathogens concerned are not in any way impeded by the flow of resin.

CHAPTER 6

THE CONTROL OF INFECTIOUS DISEASES OF PLANTS

THE control of infectious diseases of plants is effected through: (a) breeding of resistant varieties (p. 356), (b) prophylaxis against infection, (c) prophylaxis against disposition, and (d) therapy. Breeding and disposition prophylaxis represent indirect control measures and therapy a direct control measure, whilst infection prophylaxis combines both methods of approach. At present certain diseases, e.g. wart disease of potato and cereal rusts, can be countered only by genetical means, and other diseases, e.g. virus diseases of potatoes, only by continued renewal of the plant stock (i.e. by the elimination of diseased material).

§ 1. Prophylaxis against Infection

The aim of infection prophylaxis is protection before attack; it endeavours to interrupt the infection chain prior to the infection of the susceptible host. Thus it corresponds to hygiene in medicine and, like this, is preventive in character. The methods employed have been exhaustively discussed by various authors in Sorauer's *Handbuch der Pflanzenkrankheiten* (6. Band; 1939 and 1941) and we shall here restrict ourselves to certain basic principles.

Prophylaxis against infection may be attempted in two ways:

- (1) By preventing the pathogen and host from coming together, and
- (2) should this happen, by destroying the pathogen either before or during its germination.

1. Methods of rendering the meeting of host and parasite more difficult (Braun, 1937) are either indirect (prevention, points (a)–(d), (h)) or direct (active measures, points (e)–(g)).

(a) Choice of sowing time. The best time for sowing is that which encourages a vigorous growth of the young plants and a quick passage through the susceptible phase of development. The biological conditions for the infectious diseases which have to be considered are often opposed to one another (e.g. attack of wheat by bunt, p. 394, and by *Gibberella*, p. 397) and the actual course of the weather often differs from expectation and, therefore, disappointments are unavoidable. The choice of sowing time can be assisted in potatoes, for instance, by pre-sprouting.

(b) Correct sowing or planting depth. In favourable weather, shallow sowing encourages germination and thus shortens the susceptible phase of development (p. 253), whereas deep sowing has the opposite effect.

(c) Correct planting distance. Too close planting tends to raise the

atmospheric humidity in the stand and thus encourages spore production and the dispersal of parasitic fungi. Wide spacing, e.g. of vines and potatoes, improves ventilation in the stand, hastens drying of the foliage, and makes the use of the spraying machine easier.

(d) Biological decontamination of the soil. Crop rotation and regular fallowing temporarily remove susceptible hosts and, consequently, the pathogens remaining in the soil are deprived of their means of subsistence (p. 97).

(e) Physical disinfection of the soil, e.g. in horticulture, by means of superheated steam.

(f) Chemical disinfection of the soil. This method is still in the early stages of its development and, on account of high costs of material and labour, is at present applicable only in horticulture.

(g) Eradication of alternative hosts. Because of the rapidly decreasing probability of contact with increasing distance from the focus of dispersal, it is possible to break an infection chain rapidly by eradication of an alternative host, especially in infectious diseases with obligate alternation of hosts, e.g. by eradication of the juniper in the case of cluster cup rust of pear trees (p. 86).

In infectious diseases with facultative alternation of hosts, e.g. black rust of cereals, eradication of the alternate host (*Berberis* bushes) merely delays the advent of disease but does not stop it (p. 120). However, in some cases, the temporal displacement of the infection chain by a few links is enough to obviate the heaviest economic damage.

(h) Isolation measures (quarantine) by means of legislation for plant protection and regulations within any given country. Where the pathogen is only weakly propagative, e.g. wart disease of potato, the rate of dissemination may be slowed down by requiring certificates of health and origin, but obviously with highly dispersive pathogens such as downy mildew of vines and late blight of potatoes, no one can prevent the conidia from transgressing national boundaries. Hence the effectiveness of any inter-state police measures depends very largely on the biological character of the pathogen.

2. The elimination of a pathogen which has already reached the host may be achieved in one of three ways according to the biological conditions (Trappmann, 1927; Riehm, 1931; Martin, 1940).

(a) Physical disinfection of the seed and plant material; e.g. hot air or warm water treatment for loose smut of wheat and barley (p. 95).

(b) Chemical disinfection of seed and plant material, so-called 'steeps'. The chemical reagent, usually a mercury compound, may be used in liquid form (wet treatment) or as powder (dry treatment). The effect of steeping is termed primary if the fungicidal action comes into play during the steeping or drying processes, and secondary if it only develops later, after sowing. The chlorides of mercury, for instance, have a primary steeping effect on bunt spores because they kill them directly. Copper sulphate, on the contrary, has a secondary steeping effect because it is merely adsorbed

superficially by the brandspores; its toxic action develops only when germination begins; hence it does not kill the resting brandspores but hinders their germination, or destroys them in the early stages of germination. Primary steeping agents offer the greater security because, with steeping agents having secondary effects, ion exchange may take place in the soil or they may be converted into an insoluble form.

By analogy with pharmacology attempts have been made to characterize the value of chemical disinfectants by the chemo-therapeutic index, i.e. the *dosis curativa* divided by the *dosis tolerata*. The *dosis curativa* is the lowest concentration which will just suffice to render the pathogen harmless, and the *dosis tolerata* is the highest concentration which the host can support without injury. Thus the ideal disinfectant would have a very low chemo-therapeutic index: i.e. a low *dosis curativa* and a high *dosis tolerata*. In the mercury compounds used, e.g. against bunt of wheat, the index is about 0.3, and grain injury occurs only if the preparation is three times too concentrated or carelessly dissolved.

However, the value of a steeping agent cannot be defined by this index with any general validity, since the *dosis tolerata* and the *dosis toxica* sometimes vary with the variety, the quality of the crop, and the mode of storage.

(c) Chemical protection of growing plants. In some mycoses infection from the air occurs over the whole surface of a plant, or at least over all its foliage; hence the total surface of plants to be protected must be covered prophylactically with a poison. In dry air this acts in gaseous form directly on the pathogen and its spores (e.g. production of hydrogen sulphide in the layer of air next to the leaf when sulphur is dusted on vines and roses to control powdery mildew); or it may act in solution during the germination of the spores of the pathogen (e.g. copper compounds used as sprays in viticulture and fruit culture).

In all sprays two opposed requirements need to be balanced against one another: (i) the agents must be soluble in rain water or in the exudations of germ tubes, because only soluble substances are poisonous, and (ii) they must adhere in spite of their solubility, i.e. they must not be washed off, so that the reserve of salt applied may last as long as possible. The aim, therefore, is to fix the active agent to the cutin, i.e. the wax micellae, by making use of their surface activities. In order to bring about the closest possible contact between the protective agent and the leaf surface, either wetting agents which reduce the surface tension, or counteracting electric charges, are employed.

Doubtless, in the future, organic substances of far greater specific toxicity for given pathogens will replace the present non-specific protective substances (e.g. compounds of sulphur, mercury, and copper).

§ 2. Prophylaxis against Disposition

Disposition prophylaxis seeks to increase the defence readiness of the host. It becomes effective only if infection prophylaxis has failed. Its

basis was described at length in Chapter 4 ('The Disease Proneness of the Host'). The end is usually achieved in the following ways:

- (1) Breeding measures, i.e. the creation of varieties which are genetically immune or resistant to disease (Roemer *et al.*, 1938).
- (2) Agricultural and silvicultural measures; reduction of individual proneness to disease by correct choice of varieties (p. 359), soil improvement, cultivation (p. 423), suitable manuring (p. 382), &c. (Braun, 1937).

§ 3. Therapy

Therapy aims at disinfecting the diseased organism or at improving its reaction norm, so that, in ideal cases, recovery from infection takes place. Therapeutic measures in infectious diseases of plants are either (1) of a general biological nature or (2) of a physico-chemical nature.

1. The general biological measures are designed to create the most favourable setting for the reactivity of the diseased plant in order to assist the processes of localization and healing. In practical agriculture and forestry they are employed non-specifically and, as a rule, they coincide with cultural measures of disposition prophylaxis.

2. Of the physico-chemical methods, the chemical group is much the more important. But physical therapeutic measures can be used in certain infectious diseases of plants; for example, the tumours of *Bacterium tumefaciens* (Fig. 288) are particularly sensitive to X-rays and by suitable doses of radiation can be made to retrogress without injury to the adjacent healthy tissues (Rivera, 1935, 1942).

Chemotherapy, however, in contrast to its great successes in medicine and veterinary science is, at present, only of academic interest in plant pathology, being without practical application. The reasons for this are as follows:

(a) The restricted range of the chemotherapeutic index. The infectious diseases of man which interest us in this connexion are caused by pathogenic bacteria and fungi, i.e. by living plant organisms. The chemotherapeutic index, the difference between the *dosis curativa* and the *dosis toxica*, therefore comprises the whole difference between animal and plant life, and thus it is quite possible to find substances which are highly toxic to plant protoplasm but little harmful or even innocuous to human protoplasm. In infectious diseases of plants, however, higher plants are in conflict with lower plants, i.e. like with like and, hence, the therapeutic index in most cases is approximately unity. Therefore, highly specific substances must be found which will affect the cryptogamic pathogen without harming the phanerogamic host. The existence of such compounds is indicated by penicillin and similar antibiotics which, although produced by fungi, are among our most powerful antibacterial substances.

(b) The difficulty of incorporation. How can the therapeutically active material be distributed uniformly through the plant? Roughly speaking, the physician makes his patient swallow the medicine or he can introduce it

by some other means into the blood-stream. The botanist, however, has to incorporate his medicine in the soil and leave it to be taken up by the roots. But since the plasmatic membranes of plants are semipermeable, the therapeutically active substance must, in the first place, be of such a character that it can readily be taken up by the roots of the plant without undergoing any change (Krenke, 1933, pp. 841 et seq.).

(c) The slight tendency to recovery. The physician encourages in his patient the spontaneously occurring healing processes. But, in the plant body, the tendency to heal is so slight (p. 290) that the plant pathologist would have to start from an extraordinarily low level.

(d) The economic limitations. Unlike medicine, which seeks to preserve human life, plant pathology has no ideal aim but is directed to a purely economic end, the increase of profit from primary production. Its cost in chemicals and labour must, therefore, always be lower than the market value of the increased yield, as otherwise 'it does not pay'. Thus, it is not only the chemotherapeutic margin which is very narrow in the control of infectious diseases of plants, but also the economic margin. Because of the low market value of single plants (p. 3) it is out of the question for the plant pathologist to treat individuals, other than fruit trees, as the physician does. He cannot, for example, inject every wheat plant even if this were possible, but must treat the field as a whole.

The prospects for the internal therapy of plants are, therefore, not very favourable (Montemartini, 1937). Hence, at present, defence must be directed against the pathogen rather than against the disease. Plant pathology is not like individual medicine which seeks to cure every case of disease, but like group medicine (epidemiology) which seeks to interrupt the infection chain at an early stage. Its primary aim is protection from infection, and its field of work is, therefore, rightly termed plant protection, i.e. protection from infection, not plant healing. Thus, at present, it employs the methods of hygiene rather than those of medical practice.

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APPENDIX

By WILLIAM B. BRIERLEY

THE names of certain flowering plants, fungi, bacteria, &c., accepted by Professor Gäumann differ from those commonly adopted by workers in the United Kingdom or the United States. These names have been picked out and form the content of this Appendix. The names and authors as given by Professor Gäumann are followed (in parentheses) by the common names of the organism or the disease quoted from the original text; below each item are listed the more familiar synonyms or recent name changes followed (in parentheses) by the English or American common names of the organism or the disease and, on occasion, by a brief explanatory note. I am grateful to the Commonwealth Mycological Institute for checking this Appendix.

The following abbreviations are used for works cited frequently in the Appendix.

B.M.S. List.

List of Common British Plant Diseases. Compiled by the Plant Pathology Committee of the British Mycological Society. Cambridge University Press, 1944. I have also included the 'Emendations to the third edition of the List of Common British Plant Diseases' given in *Trans. Brit. mycol. Soc.*, **31**, pp. 340-2, 1948.

Cartwright & Findlay, 1946.

Decay of Timber and its Prevention. By K. St. G. Cartwright & W. P. K. Findlay. London: H.M. Stationery Office, 1946.

Holmes, *Hb.*

Handbook of Phytopathogenic Viruses. By F. O. Holmes. Minneapolis, Minn.: Burgess Publishing Co., 1939.

M.D.B., 1948.

Bergey's Manual of Determinative Bacteriology. By R. S. Breed, E. G. D. Murray, A. Parker Hitchins, and sixty other contributors. London: Baillière, Tindall, & Cox. 6th edition, 1948.

R.A.M., 1946.

'Common names of virus diseases used in the Review of Applied Mycology', *Rev. appl. Mycol.*, **24**, special part 13, 1946.

Smith, *Tb.*

A Textbook of Plant Virus Diseases. By K. M. Smith. London: J. & A. Churchill Ltd., 1937.

S.P.N.

Standardized Plant Names: a Revised and Enlarged Listing of Approved Scientific and Common Names of Plants and Plant Products in American Commerce or Use. Prepared for the American Joint Committee on Horticultural Nomenclature by its Editorial Committee, Harlan P. Kelsey and William C. Dayton. Harrisburg, Pa.: J. Horace McFarland Co. 2nd edition, 1942.

The names of *Fusarium* spp. have been checked with (i) *Die Fusarien.* By H. W. Wollenweber & O. A. Reinking. Berlin: Paul Parey, 1935; and (ii) W. C. Snyder & H. N. Hansen, *Amer. J. Bot.*, **27**, pp. 64-7, 1940; *ibid.*, **28**, pp. 738-42, 1941; *ibid.*, **32**, pp. 657-66, 1945.

The names of all fungi, bacteria, viruses, &c., have been checked with the usage of the Commonwealth Mycological Institute as exemplified in the *Review of Applied Mycology*.

The names of flowering plants have been checked with various standard *Floras* and other works on systematic botany.

'*Abies pectinata* DC.'; ('Weisstanne', 'Tanne').

= *A. alba* Mill.; (European silver fir (*S.P.N.*); common silver fir).

Abutilon mosaic; ('Abutilon-Mosaik').

Abutilon variegation virus (*R.A.M.*, 1946); *Abutilon* virus 1 (Smith, *Tb.*); *Marmor abutilon* (*M.D.B.*, 1948).

'*Abutilon Thompsoni* André'

= *A. striatum* Dicks. var. *thompsoni* Veitch; (red-vein abutilon (*S.P.N.*)).

'*Agaricus adiposus* Fr.'; ('Muschelbruch').

= *Pholiota adiposa* Fr.; (fatty pholiota (*S.P.N.*); yellow cap fungus, greasy scaly cap). Causes a brown mottled rot of the heart-wood of living hardwoods and certain conifers.

'*Albugo candida* (Pers.) Ktze.'; ('Weissrost').

= *Cystopus candidus* (Pers. ex Chev.) Lév.; (white blister of crucifers (*B.M.S. List*)).

Alpenrose; ('Alpenrosen').

Rhododendron ferrugineum L.; (rock rhododendron (*S.P.N.*)); and *R. hirsutum* L.; (garland rhododendron (*S.P.N.*)).

'*Ampelopsis Veitchii* Ht.'; ('Jungfernrebe').

= *Parthenocissus tricuspidata* var. *veitchii* Planch.; (Veitch's Japanese creeper (*S.P.N.*); Virginian creeper).

'*Aplanobacter michiganense* E.F.S.'

See under *Bacterium michiganense*.

'*Arrhenatherum elatius* (L.) M. & K.'; ('... französischen Raygras').

= *A. avenaceum* Beauv.; (tall oat-grass (*S.P.N.*); false oat-grass).

Aster mosaic; ('Astermosaik').

Aster yellows virus (*R.A.M.*, 1946); *Callistephus* virus 1 (Smith, *Tb.*); *Chlorogenus callistephi* (*M.D.B.*, 1948).

Aucuba mosaic (of potato).

See Potato aucuba mosaic.

'*Bacillus amylovorus* (Burr.) Trev.'; ('... Feuerbrandes der Obstbäume').

= *Ercinia amylovora* (Burr.) Winslow *et al.* (*M.D.B.*, 1948); syn. *Bacterium amylovorum* (Burr.) Chester; (fire blight of apple, pear, &c.).

'*Bacillus asterosporus* (Mey.) Mig.'

= *B. polymyxa* (Prazmowski) Mig. (*M.D.B.*, 1948).

'*Bacillus atrosepticus* van Hall'; (*orig.*, p. 74: '*Bacillus atrosepticus* van Hall (syn. *Bac. phytophthorus* Appel), der Erreger der Schwarzbeinigkeit der Kartoffelstauden (Abb. 298) und einer Nassfäule der Knollen'; p. 329: 'Die bakteriellen Erreger der Nassfäulen, so *Bacillus atrosepticus* (Schwarzbeinigkeit) und *Bacillus carotovorus* Jones (Weichfäule der Möhren, auch auf Kartoffeln übergehend)'; p. 387: '... *Bacillus carotovorus* Jones (Rübenfäule) und *Bacillus atrosepticus* van Hall (Schwarzbeinigkeit der Kartoffeln) . . .'; pp. 455, 482, 541, 548: '... *Bacillus atrosepticus* van Hall . . .'; pp. 522, 531: '*Bacillus atrosepticus* der Erreger der Schwarzbeinigkeit der Kartoffelstauden (Abb. 298)'; p. 547, subscript to Fig. (Abb. 298) of potato black leg (after

Stapp, 1932): 'Schwarzbeinigkeit der Kartoffelstauden, verursacht durch *Bacillus atrosepticus* v. Hall').

The relationships of the *carotovorum*-*phytophthorum* bacteria are obscure and controversial. Gäumann apparently follows Stapp in regarding *Bacillus atrosepticus* and *Bacillus carotovorus* as strains of *Bacterium phytophthorum*; *Bacillus atrosepticus* causing potato black leg and a wet rot of potato tubers, and *Bacillus carotovorus* causing a soft rot of vegetables.

English usage (*B.M.S. List*) is as follows: (i) black leg of potato—*Bacterium phytophthorum* (Appel) Burgwitz; syn. *Erwinia phytophthora* (Appel) Bergey *et al.*: (syn. *Bacillus phytophthorus* Appel); (ii) soft rot of vegetables—*Bacterium carotovorum* (L. R. Jones) Lehm.; syn. *Erwinia carotovora* (L. R. Jones) Holland; (syn. *Bacillus carotovorus* Jones).

American usage (*M.D.B.*, 1948) is as follows: (i) black leg of potato—*Erwinia atroseptica* (van Hall) Jennison; syn. *Bacillus atrosepticus* van Hall; (ii) soft rot of vegetables—*Erwinia carotovora* (Jones) Holland; syn. *Bacillus carotovorus* Jones; (syn. *Bacterium carotovorum* Lehm. & Neum.).

'*Bacillus botulinus* van Erm.'; ('Wurstvergiftungen').

= *Clostridium botulinum* (van Erm.) Holland (*M.D.B.*, 1948); (botulism or bad-meat poisoning).

'*Bacillus carotovorus* Jones'

See under *B. atrosepticus*.

'*Bacillus proteus* Trev.'

= *Proteus vulgaris* Hauser (*M.D.B.*, 1948).

'*Bacillus radiobacter* Beij. & van Deld.'

= *Agrobacterium radiobacter* (Beij. & van Deld.) Conn (*M.D.B.*, 1948); syn. *Alcaligenes radiobacter* Conn; syn. *Rhizobium radiobacter* Pribram.

'*Bacillus Savastanoi* E.F.S.'; ('Tuberkeln des Ölbaumes'; 'Ölbaumtumore').

= *Pseudomonas savastanoi* (E.F.Sm.) Stevens (*M.D.B.*, 1948); syn. *Phytomonas savastanoi* (E.F.Sm.) Bergey *et al.*; (knot, tubercle, or gall of the olive tree).

'*Bacillus suispestifer* Kruse'

= *Salmonella choleraesuis* (Smith) Weldin (*M.D.B.*, 1948); (secondary invader in swine fever or hog cholera).

'*Bacillus tetani* Nic.'; ('... Wundstarrkrampfes').

= *Clostridium tetani* (Flügge) Holland (*M.D.B.*, 1948); (tetanus or lock-jaw).

'*Bacillus vulgatus* Trev.'; (*orig.*, p. 429: 'Gewisse Erreger von Nassfäulen bei Kartoffelknollen, so *Bacillus vulgatus* Trev. und *Bacillus subtilis* Cohn . . .').

B. vulgatus is probably identical with or a variant of *B. subtilis* Cohn emend. Prazmowski (*M.D.B.*, 1948); (often called the potato bacillus).

'*Bacterium angulatum* Fromme & Murray'; ('... eckigen Blattfleckenkrankheit des Tabaks').

= *Pseudomonas angulata* (Fromme & Murray) Holland (*M.D.B.*, 1948); syn. *Phytomonas angulata* (Fromme & Murray) Bergey *et al.*; (angular leaf spot or black fire of tobacco).

See also under *Bacterium tabacum*.

'*Bacterium coli* Esch.'

= *Escherichia coli* (Mig.) Castellani & Chalmers (*M.D.B.*, 1948); (a normal inhabitant of the intestine of man and all vertebrates; often causes infections of the genito-urinary tract).

'*Bacterium delphinii* E.F.S.'; ('Schwarzfleckigkeit des Ritterspornes').

= *Pseudomonas delphinii* (E.F.Sm.) Stapp (*M.D.B.*, 1948) (*B.M.S. List*); syn.

Phytomonas delphinii (E.F.Sm.) Bergey *et al.*; (black blotch of delphinium and larkspur (*B.M.S. List*)).

'*Bacterium exitiosum* Gard. & Kend.'

See *B. vesicatorium*.

'*Bacterium herbicola aureum* Dügg.'

? = *Pseudomonas trifolii* Huss (*M.D.B.*, 1948).

'*Bacterium maculicola* McCull.'; ('Blattfleckenkrankheit des Blumenkohls').

= *Pseudomonas maculicola* (McCull.) Stev. (*M.D.B.*, 1948) (*B.M.S. List*); syn. *Phytomonas maculicola* Bergey *et al.*; syn. *Bacterium maccullochianum* Burgwitz; (bacterial leaf spot of cabbage, cauliflower, &c. (*B.M.S. List*)).

'*Bacterium malvacearum* E.F.S.'; ('...eckigen Blattfleckenkrankheit der Baumwollsträucher').

= *Xanthomonas malvacearum* (E.F.Sm.) Dowson (*M.D.B.*, 1948); syn. *Phytomonas malvacearum* (E.F.Sm.) Bergey *et al.*; syn. *Pseudomonas malvacearum* E.F.Sm.; (angular leaf spot of cotton).

'*Bacterium medicaginis* (Sack.) E.F.S. var. *phaseolicola* (Burkh.) Lk. & Hall'; ('Fettfleckenkrankheit der Bohnen').

= *Pseudomonas phaseolicola* (Burkh.) Dowson (*M.D.B.*, 1948) (*B.M.S. List*); syn. *Phytomonas medicaginis* var. *phaseolicola* Burkh.; (halo blight of bean (*B.M.S. List*)).

'*Bacterium michiganense* E.F.S.'

Gäumann speaks of *Bacterium michiganense* (orig., p. 164) as 'der Erreger der bakteriellen Tomatenwelke', and of *Aplanobacter michiganense* E.F.S. (orig., p. 322) as the pathogen of '... Bakterienkrebs' of tomato. The names are obligate synonyms.

= *Corynebacterium michiganense* (E.F.Sm.) H.L.Jens. (*M.D.B.*, 1948) (*B.M.S. List*); syn. *Aplanobacter michiganense* E.F.Sm.; syn. *Bacterium michiganense* E.F.Sm.; syn. *Pseudomonas michiganensis* (E.F.Sm.) Stevens; syn. *Phytomonas michiganensis* (E.F.Sm.) Bergey *et al.*; (bacterial canker of tomato (*B.M.S. List*); Grand Rapids disease of tomato (U.S.A.)).

'*Bacterium ozaenae* Lehm. & Neum.'; ('der vermutliche Erreger der Stinknase').

? = *Klebsiella ozaenae* (Abel) Bergey *et al.*, which is possibly only a variety of *Klebsiella pneumoniae* (Schroeter) Trevisan (*M.D.B.*, 1948); (stink-nose or foetid nasal catarrh).

'*Bacterium pneumoniae*'; ('Lungenentzündung').

Gäumann appends no author to this name which is thus ambiguous. *Bacterium pneumoniae* Lehm. & Neum. is Friedländer's pneumobacillus, *Klebsiella pneumoniae* (Schroeter) Trevisan, whereas *Bacterium pneumoniae* Mig. is the pneumococcus, *Diplococcus pneumoniae* Weichselb.; (see *M.D.B.*, 1948).

'*Bacterium prodigiosum* Lehm. & Neum.'; ('Rotfärbung stärkereicher Substrate').

= *Serratia marcescens* Bizio; syn. *Micrococcus prodigiosus* Cohn; syn. *Chromobacterium prodigiosum* Topley & Wilson; (*M.D.B.*, 1948).

'*Bacterium putidum* (Flügge) Lehm. & Neum.'

= *Pseudomonas putida* (Trevisan) Migula; (*M.D.B.*, 1948).

'*Bacterium radicicola* Beij.'; ('Wurzelknöllchenbakterien der Leguminosen').

= *Rhizobium leguminosarum* Frank emend. Baldwin & Fred (*M.D.B.*, 1948); syn. *Rhizobium radicicola* Hilt. & Störm.; syn. *Pseudomonas radicicola* Moore; (root nodule bacterium, all species).

'*Bacterium sepedonicum* Spieck & Kotth.'; ('Bakterienringfäule der Kartoffeln').

= *Corynebacterium sepedonicum* (Spieck. & Kotth.) Skaptason & Burkholder

(*M.D.B.*, 1948); syn. *Aplanobacter sepedonicum* E.F.Sm.; syn. *Phytomonas sepedonica* Magrou; (bacterial ring rot of potato).

'*Bacterium solanacearum* E.F.S.'; ('Schleimkrankheit mancher Solanaceen').

= *Pseudomonas solanacearum* E.F.Sm. (*M.D.B.*, 1948); syn. *Xanthomonas solanacearum* (E.F.Sm.) Dowson; syn. *Phytomonas solanacearum* (E.F.Sm.) Bergey *et al.*; (brown rot of Solanaceae).

'*Bacterium syringae* (van Hall) E.F.S.'; ('Zweigkrankheit des Flieders, usw.').

= *Pseudomonas syringae* van Hall (*M.D.B.*, 1948) (*B.M.S. List*); syn. *Phytomonas syringae* (van Hall) Bergey; (bacterial blight of lilac (*B.M.S. List*)).

See also under *Pseudomonas cerasi*.

'*Bacterium tabacum* Wolf & Foster'; ('Wildfeuerkrankheit des Tabaks').

Gäumann cites this organism (*orig.*, pp. 30, 259, 322, 376) by the above name; on p. 463 (*orig.*) he cites it by the name *Pseudomonas tabaci* without author, following Böning (1930) to whose work he is referring.

= *Pseudomonas tabaci* (Wolf & Foster) Stevens (*M.D.B.*, 1948); syn. *Phytomonas tabaci* (Wolf & Foster) Bergey *et al.*; (wildfire of tobacco).

On p. 30 (*orig.*) Gäumann cites *Bacterium angulatum* Fromme & Murray, the pathogen of angular leaf spot of tobacco ('... eckigen Blattfleckenkrankheit des Tabaks').

= *Pseudomonas angulata* (Fromme & Murray) Holland (*M.D.B.*, 1948); syn. *Phytomonas angulata* (Fromme & Murray) Bergey *et al.*; (angular leaf spot or black fire of tobacco).

According to Stapp (1930) and Braun (1937), *Pseudomonas tabaci* and *Ps. angulata* are identical in culture but differ in the type of disease they produce. Braun found that the symptoms produced by non-toxic strains of *Ps. tabaci* are indistinguishable from those produced by *Ps. angulata* but does not consider it desirable to combine the two organisms under a single name as is done by Stapp. The distinction is maintained by Dowson (1943) and in *M.D.B.*, 1948.

'*Bacterium tumefaciens* Sm. & Towns.'; ('... bakteriellen Pflanzenkrebses').

= *Agrobacterium tumefaciens* (Smith & Townsend) Conn (*M.D.B.*, 1948); syn. *Phytomonas tumefaciens* (Sm. & Towns.) Bergey *et al.*; (crown gall).

'*Bacterium typhi abdominalis* Flügge.'

= *Salmonella typhosa* (Zopf) White (*M.D.B.*, 1948); syn. *Bacillus typhosus* Zopf; syn. *Eberthus typhosus* Castellani & Chalmers; syn. *Eberthella typhosa* Weldin; (typhoid fever).

'*Bacterium vesicatorium* Doidge (syn. *Bacterium exitiosum* Gard. & Kend.)'; ('der Erreger einer "Krebs" genannten Fleckenkrankheit der Tomaten, Kartoffeln, usw.').

= *Xanthomonas vesicatoria* (Doidge) Dowson (*M.D.B.*, 1948); syn. *Phytomonas vesicatoria* Bergey *et al.*; syn. *Pseudomonas vesicatoria* Stapp; (bacterial spot of tomato and other Solanaceae).

Bean mosaic; ('Bohnenmosaik').

Bean mosaic virus (*R.A.M.*, 1946); *Phaseolus virus* 1 (Smith, *Tb.*); *Marmor phaseoli* (*M.D.B.*, 1948).

Beet curly top; ('Kräuselkrankheit der Zuckerrüben').

Beet curly top virus (*R.A.M.*, 1946); *Beta virus* 1 (Smith, *Tb.*); *Ruga verrucosans* Carsner & Bennett (*M.D.B.*, 1948), syn. *Chlorogenus eutetticola* (Holmes, *Hb.*).

Beet mosaic; ('Zuckerrübenmosaik').

Beet mosaic virus (*R.A.M.*, 1946); *Beta virus* 2 (Smith, *Tb.*); *Marmor betae* (*M.D.B.*, 1948).

Beet phloem necrosis; ('Phloëmnekrose bei Zuckerrüben').

See Beet curly top.

Bromus spp.; ('Trespen').

In referring to investigations by Salmon (1904) and by Hammarlund (1925), Gäumann cites (*orig.*, pp. 266-7) three species of *Bromus*: (i) *B. racemosus* L.—'... traubigen Trespe', (ii) *B. pratensis* Ehrh. (syn. *B. commutatus* Schrad.)—'Wiesentrespe', and (iii) *B. hordeaceus* L.—'Gerstentrespe'.

Salmon (1904), who does not append authors to his names, states that his *B. hordeaceus* is 'identical morphologically with *B. mollis*' but that it possesses 'different constitutional characters', and that his *B. commutatus* and *B. racemosus* 'which resemble each other so closely morphologically that most systematists rank them as a species and its variety', possess distinctive physiological (or constitutional) characters'. Hammarlund (1925) uses the names *B. racemosus*, *B. commutatus*, and *B. hordeaceus* without authors or comment.

It is difficult to know what value, in terms of scientific experimentation or logical induction, to attach to these names since the relationships and nomenclature of *Bromus* spp., and especially of the forms included in the section *Serrafalcus*, are almost hopelessly confused. Thus, perhaps most authors (e.g. Ascherson & Graebner, Schinz & Keller, Hegi, Armstrong, &c.) recognize *B. racemosus*, *B. commutatus* (= *pratensis*), and *B. mollis* (= *hordeaceus*) as good species; some authors (e.g. Bentham & Hooker, Marshall Ward, &c.) reduce them all to varieties of *B. arvensis* L.; Coste recognizes *B. mollis* and *B. hordeaceus* as distinct species and this position seems to be accepted by many American workers (e.g. Hitchcock, Silveus, &c.), who state that whilst *B. mollis* is a common weed, *B. hordeaceus* is 'a distinct European species' which is 'not known in America'; Hooker, Druce, &c., recognize *B. mollis* and *B. racemosus* with its variety *commutatus*; Clapham (1946) lists *B. racemosus* L., *B. commutatus* Schrad. (*B. pratensis* Ehrh.), and tentatively *B. mollis* L. sec. Holmbg. (*B. hordeaceus* auct.) and *B. hordeaceus* L. sec. Holmbg. (*B. thominii* Hard.).

Recent cytogenetic investigations have shown the genus *Bromus* to contain a polyploid series with $x = 7$ (see Darlington & Ammal, 1945); *B. arvensis* being diploid, *B. hordeaceus* (*mollis*) and *B. racemosus* tetraploid, and *B. commutatus* octaploid. Further, it is now known that extensive intergeneric and interspecific hybridization occurs in *Bromus* with resultant innumerable segregations. These conditions, plus the morphological and physiological differentiation of ecotypes in various geographic regions and under different environmental conditions in any one region, would produce the vast swarm of apparently intergrading morphological forms and physiological races which is presented by the section *Serrafalcus* of the genus *Bromus*. It would seem that the specific names, *mollis*, *hordeaceus*, *racemosus*, *commutatus*, &c., merely distinguish arbitrary peripheral morphological forms of this swarm and that the morphology of individual plants may bear no constant or inherent relationship to their physiology, e.g. their disease resistance. This, indeed, is indicated by Salmon (1904), who states: 'It would appear then that "races" of the species *B. commutatus* exist which possess distinctive physiological characters as shown by their different reaction to the same fungus. Evidence of the same nature has been obtained with regard to other host-species.'

In view of the above situation it is clear that little if any value can be attached to the names of brome grasses cited in Gäumann's original text or to conclusions based upon the earlier investigations which themselves depend upon specific identifications of brome grasses within the section *Serrafalcus*.

The most common vernacular names given to the forms mentioned are: *B. racemosus*—Trauben-Trespe (Hegi), bald brome (*S.P.N.*), smooth brome

grass, upright chess; *B. commutatus* (*pratensis*)—Verwechselte Trespe (Hegi), hairy brome (*S.P.N.*), but Muenscher (1935) and many English authors give smooth brome grass; *B. mollis* (*hordeaceus*)—Weiche Trespe (Hegi), soft brome grass, soft chess.

'*Bryophyllum crenatum* Baker.'

= *Kalanchoë laxiflora* Baker.

'*Cereus speciosus* (Cav.) Schum. (syn. *C. spectabilis*).'

= *Heliocereus speciosus* Schum.

'*Cicadula sexnotata* Fall.'; ('Zwergzikade').

Smith (*Hb.*) states '*Cicadula sexnotata* Fall., long known as the vector of *Callistephus virus 1* (aster yellows virus), is now said to be a European species whilst the name of the vector, which is only found in North America, should be *Cicadula divisa* Uhler, and this has recently been changed to *Macrosteles divisus* Uhler'. Holmes (*M.D.B.*, 1948) gives '*Macrosteles divisus* (Uhl.) (= *Cicadula sexnotata* (Fall.), *C. divisa* (Uhl.))'.

'*Citrus grandis* Hassk.'; ('Grapefruit-Sorten').

= *C. maxima* Merr.; (pummelo (*S.P.N.*)).

'*Coniophora cerebella* (Pers.) Schroet.'; ('Trockenfäule').

= *C. puteana* (Schum.) Karst.; (cellar fungus). Causes a brown cubical rot of conifers and occasionally of hardwoods, but is of most importance as a destroyer of timber and in buildings is perhaps the commonest agent of decay in damp wood. (See Cartwright & Findlay, 1946.)

'*Corticium vagum* B. & C. (syn. *Rhizoctonia solani* Kuhn)'; ('Wurzeltöter der Kartoffeln'; 'Filzkrankheit der Kartoffelstengel'; 'Pocken der Kartoffelknollen'; 'Wurzelbrand der Zuckerrüben- und Koniferenkeimlinge').

= *Corticium solani* (Prill. & Delacr.) Bourd. & Galz., stat. mycel. *Rhizoctonia solani* Kühn; (black leg of beet, black scurf and stem canker of potato (*B.M.S. List*)). Moore (*Diseases of Crop Plants*, 1943-6, Minist. Agric. Bull., No. 139, 1948) gives also sharp eyespot of wheat; root rot of barley, blackberry, &c.; wire stem of tomato, kale, &c.; foot rot of pea; damping-off of seedlings; canker of radish; rusty root of celery; root and stem base rot of flax seedlings, &c.

Crinkle (of potato).

See Potato crinkle.

Cucumber mosaic; ('Gurkenmosaik III und IV').

III. Cucumber green-mottle mosaic virus (*R.A.M.*, 1946); *Cucumis virus 2* (Smith, *Tb.*); *Marmor astrictum* (*M.D.B.*, 1948).

IV. A distinctive strain of the above virus. Cucumber yellow mosaic virus (*R.A.M.*, 1946); *Cucumis virus 2A* (Smith, *Tb.*); *Marmor astrictum* var. *aucuba* (*M.D.B.*, 1948).

Curly top (of beet).

See Beet curly top.

'*Datura Tatula* L.'

A purple-flowered variant of *D. stramonium* L.; (jimsonweed (*S.P.N.*); purple thorn apple).

'*Diplachne serotina* (L.) Link.'

= *Cleistogenes serotina* (L.) Keng.

'*Diplocarpon rosae* (Lib.) Wolf'; ('Sternrusstau der Rosen').

Stat. conid. *Actinonema rosae* (Lib.) Fr.; (black spot of rose (*B.M.S. List*)). Baker (*Plant Dis. Rept.*, 32 (6), pp. 260-74, 1948) considers *Marssonina rosae* (Lib.) Lind to be the correct binomial for the imperfect state, and finds that

the perfect state, *Diplocarpon rosae* Wolf, has been recorded only from the United States and Canada.

'*Discula pinicola* (Naoum.) Petr.'; ('Blaufäule des Nadelholzes').

= *Gloeosporium pinicola* (Naoum.) Petr., Clem. & Shear; (blue stain in wood).

'*Elymus europaeus* L.'; ('Haargras').

= *Hordeum europaeum* (L.) All.; syn. *H. sylvaticum* Huds.; (wood barley grass).

'*Endostigme inaequalis* (Cke.) Syd.'; ('Apfelschorf').

= *Venturia inaequalis* (Cooke) Wint., stat. conid. *Fusicladium dendriticum* (Wallr.) Fuckel; (apple scab).

Sydow was of opinion (*Ann. mycol., Berl.*, **21**, p. 171, 1923) that this fungus does not properly belong to the genus *Venturia* and transferred it to his new genus *Endostigme* as *E. inaequalis* (Cke.) Syd. The name *Endostigme* has been accepted by certain Continental authors but is not used in the United Kingdom and the United States. For a discussion of the situation see Jørstad, I., 'What is the correct botanical name of the apple scab fungus?' (*Nyt. Mag. Naturv.*, **84**, p. 251, 1943).

'*Endostigme pirina* (Aderh.) Syd.'; ('Birnschorf').

= *Venturia pirina* Aderh., stat. conid. *Fusicladium pirinum* (Lib.) Fuckel; (pear scab). See note under *Endostigme inaequalis*.

F virus (of potato).

See Potato aucuba mosaic.

'Föhn.'

A warm dry south wind that blows down the valleys on the north side of the Alps in Switzerland.

'*Fusarium avenaceum* (Fr.) Sacc.'

See *F. herbarum*.

'*Fusarium bulbigenum* C. & M.'; ('Wurzelbrand der Fichte').

= *F. oxysporum* Schl. ex Fr.; (root rot of spruce).

'*Fusarium bulbigenum* C. & M. var. *batatas* Woll.'; ('Batatenwelke').

= *F. oxysporum* Schl. ex Fr., f. *batatas* (Wollenw.) Snyder & Hansen; (wilt of sweet potato).

'*Fusarium cepae* Hanz.'

= *F. oxysporum* Schl. ex Fr., f. 7 Wollenw.; syn. *F. oxysporum* Schl. ex Fr., f. *cepae* (Hanz.) Snyder & Hansen.

'*Fusarium conglomerans* Woll.'; ('... nordamerikanischen Kohlwelke').

= *F. oxysporum* Schl. ex Fr., f. *conglomerans* (Wollenw.) Snyder & Hansen; (wilt or yellows of cabbage).

'*Fusarium cubense* E.F.S.'; ('Panamakrankheit der Bananen').

= *F. oxysporum* Schl. ex Fr., v. *cubense* (E.F.Sm.) Wollenw. & Reink.; syn. *F. oxysporum* Schl. ex Fr., f. *cubense* (E.F.Sm.) Snyder & Hansen; (Panama disease of banana).

'*Fusarium culmorum* (W.G.Sm.) Sacc.'; ('Fusariose des Roggens'; 'Ährenfusariose'; 'Schneeschimmel'; 'Keimlingskrankheit bei Weizen').

= *F. roseum* Lk., f. *cerealis* (Cke.) Snyder & Hansen; (brown foot rot and ear blight of cereals (*B.M.S. List*)).

'*Fusarium fructigenum* Fr.'; ('Knospen- und Fruchtfäule der Äpfel').

= *F. lateritium* (Nees) Snyder & Hansen; (bud rot of apple and pear (*B.M.S. List*)).

- '*Fusarium herbarum* (Cda.) Fr. (= *F. putrefaciens* Osterw.); ('Fäulniserreger des Lagerobstes').
= *F. avenaceum* (Fr.) Sacc.; syn. *F. roseum* (Lk.) Snyder & Hansen; (brown foot rot and ear blight of cereals (*B.M.S. List*)).
- '*Fusarium lini* Boll.'; ('Flachswelke').
= *F. oxysporum* Schl. ex Fr., f. *lini* (Bolley) Snyder & Hansen; (flax wilt).
- '*Fusarium lycopersici* Sacc.'; ('Tomatenwelke').
= *F. bulbigenum* Cke. & Mass., v. *lycopersici* (Brushi) Wollenw. & Reink.; syn. *F. oxysporum* Schl. ex Fr., f. *lycopersici* (Sacc.) Snyder & Hansen; (*Fusarium* wilt of tomato (*B.M.S. List*)).
- '*Fusarium Martii phaseoli* Burk.'; ('Wurzelfäule der Bohnen'; 'Welkekrankheit der Bohnen').
= *F. solani* (Mart.) v. *martii* (Appel & Wollenw. sub specie) Wollenw. forma 3 Snyder; syn. *F. solani* (Mart.) Appel & Wollenw., f. *phaseoli* (Burk.) Snyder & Hansen; (foot rot of bean (*B.M.S. List*)).
- '*Fusarium moniliforme* Sheld.'; ('Wurzelbrand mancher Keimlinge').
Imperfect state of *Gibberella fujikuroi* (Saw.) Wollenw.
- '*Fusarium nivale* (Fr.) Ces.'; ('... der (weisse) Schneeschimmel des Wintergetreides'; 'Schneeschimmel des Getreides').
= *F. nivale* (Fr.) Ces., f. *graminicola* (Berk. & Br.) Snyder & Hansen. Imperfect state of *Calonectria graminicola* Wollenw. = *G. nivalis* Schaffnit, (*B.M.S. List*). (Snow mould of turf, grasses, &c. (*B.M.S. List*)).
- '*Fusarium orthoceras* App. & Wr. var. *pisi* Linf.'; ('Welkekrankheit bei Erbsen').
= *F. oxysporum* Schl. ex Fr., f. *pisi* (Lindf.) Snyder & Hansen race 1; (wilt of pea).
- '*Fusarium putrefaciens* Osterw.'
See *F. herbarum*.
- '*Fusarium radiculicola* Woll.'; ('Trockenfäule auf Kartoffelknollen').
= *F. javanicum* Koord. v. *radiculicola* Wollenw.; syn. *F. solani* (Mart.) Appel & Wollenw., f. *radiculicola* (Wollenw.) Snyder & Hansen; syn. *Cylindrocarpon radiculicola* Wollenw. (in *R.A.M.*); (dry rot of potato tubers).
- '*Fusarium solani* (Mart.) App. & Wr.'; ('Weissfäule der Kartoffelknollen').
Imperfect state of *Hypomyces solani* Rke. & Berth.; (soft rot of potato tubers).
- '*Fusarium vasinfectum* Atk.'; ('Welkekrankheit der Baumwollsträucher'; '... nord-amerikanischen Baumwollwelke').
= *F. vasinfectum* Atk., f. 1 Wollenw.; syn. *F. oxysporum* Schl. ex Fr., f. *vasinfectum* (Atk.) Snyder & Hansen; (cotton wilt).
- '*Gibberella Fujikuroi* (Saw.) Woll.'
G. moniliformis (Sheld.) Snyder & Hansen is a synonym.
- '*Gibberella Saubinetii* (Mont.) Sacc.'; ('... Keimlingskrankheit bei Weizen und Mais'; 'Schneeschimmel des Weizens'; 'Fusskrankheit des Weizens').
= *G. zeae* (Schw.) Petch (in *B.M.S. List*); syn. *G. roseum* Link ex Fr., f. *cereale* (Cke.) Snyder & Hansen; (brown foot rot and ear blight of cereals (*B.M.S. List*)).
- '*Helminthosporium avenae* Eidam'; ('Streifenkrankheit des Hafers').
Conidial state of *Pyrenophora avenae* Ito & Kuribay.; (leaf spot and seedling blight of oats (*B.M.S. List*)).
- Hyoscyamus* III virus; ('*Hyoscyamus*-III-Virus').
Henbane mosaic virus (*R.A.M.*, 1946); *Hyoscyamus* virus 1 (Smith, *Tb.*); *Marmor hyoscyami* (*M.D.B.*, 1948).

'*Lentinus squamosus* Schroet.'

— *L. lepideus* Fr.; (scaly *Lentinus* (*S.P.N.*); scaly cap). Causes a brown cubical rot of coniferous woods, especially worked timber in contact with the soil. (See Cartwright & Findlay, 1946.)

Leucostoma (*cincta*, *nivea*); ('Apoplexie der Aprikosen-, Kirschbäume, usw.').

= *Valsa* (*cincta*, *nivea*); (die back of apricot, cherry, &c.).

'*Loroglossum* (*Himantoglossum*) *hircinum* (L.) Rich.'

Syn. *Orchis hircina* (L.) Crantz; (lizard orchid).

'*Macrosporium* *tomato* Cke.'; ('Fleckenkrankheit und Fruchtfäule der Tomaten').

— *Alternaria* *tomato* (Cooke) Weber; (leaf spot and fruit rot of tomato; nail-head spot (U.S.A.)).

Maize streak; ('Streifenmosaik des Mais').

Maize streak virus (*R.A.M.*, 1946); *Zea* virus 2 (Smith, *Tb.*); *Fractilinea maidis* (*M.D.B.*, 1948), syn. *Marmor maidis* (Holmes, *Hb.*).

'*Melampsora* *liniperda* (Koern.) Palm'; ('Flachsrost').

= *M. lini* (Ehrenb.) Lév.; (flax rust).

'*Microsphaera* *alphitoides* Griff. & Maubl.'; ('Eichenmehltau').

Stat. conid. *Oidium quercinum* Thum.; syn. *Microsphaera quercina* (Schw.) Burr.; (oak mildew). Cleistocarps first recorded in England in 1945 (*Trans. Brit. mycol. Soc.*, **29**, p. 219, 1946) and again in 1948 (*Nature, Lond.*, **161**, p. 938, 1948).

Mosaic (of potato).

See Potato mottle.

'*Mucor* *stolonifer* Ehrenb.'

— *Rhizopus stolonifer* (Ehrenb. ex Fr.) Lind; syn. *R. nigricans* Ehrenb.

Myzodes (*circumflexus*, *persicae*, *pseudosolani*).

= *Myzus* (*circumflexus*, *persicae*, *pseudosolani*).

'*Nectria* *ditissima* Tul.'; ('Buchenkrebs').

= *N. punicea* (K. & Schm.) Fr.; (beech canker).

'*Nectria* *galligena* Bres.'; ('... Zweigkrebses der Apfelbäume'; '... Apfelkrebses').

= *Dialonectria galligena* (Bres.) Petch; (canker and eye rot of apple (*B.M.S. List*)).

'*Odostemon* *repens* (Lindl.) Rydb.'

= *Mahonia repens* (Lindl.) G. Don.; (creeping Mahonia (*S.P.N.*)).

Ophiostoma (*coerulea*, *fimbriatum*, *pini*, *ulmi*).

— *Ceratostomella* (*caerulea*, *fimbriata*, *pini*, *ulmi*). *C. ulmi* Buism.; stat. conid. *Graphium ulmi* Schwarz; ('... Ulmensterbens' = Dutch elm disease).

'*Penicillium* *stoloniferum* Thom.'

= *P. brevi-compactum* Dierckx.

'*Peronospora* *brassicae* Gm.'

— *P. parasitica* (Fr.) Tul.; strain. *P. parasitica* is a group species containing numerous varieties or races some of which have been given distinctive names; see E. Gäumann, *Die Gattung Peronospora* (*Beitr. Kryptogamenfl. Schweiz*, **5** (4), pp. 1–360, 1923).

'*Peronospora* *cheiranthi* Gm.'; ('... falschen Mehltau des Goldlack').

= *P. parasitica* (Fr.) Tul.; strain. (Downy mildew of wallflower (*B.M.S. List*)). See note under *P. brassicae*.

Phloem necrosis (of beet).

See Beet curly top.

Phloem necrosis (of potato).

See Potato leaf roll.

'*Phomopsis citri* Fawc.'

Imperfect state of *Diaporthe citri* (Fawc.) Wolf.

'*Picea excelsa* (Lam.) Link'; ('Fichte').

= *P. abies* Karsten; (Norway spruce (*S.P.N.*); common spruce).

'*Pinus austriaca* Höss.'; ('Schwarzföhre').

= *P. nigra* Arnold; (Austrian pine (*S.P.N.*)). A variable species with several geographic forms or varieties of which *P. austriaca* is identical with the type.

'*Pinus montana* Mill.'; ('Legföhre'; '... aufrechten Bergföhre').

= *P. mugo* Turra.; (Swiss mountain pine (*S.P.N.*)).

Pleurage (*curvicolla*, *fimiseda*).

= *Sordaria* (*curvicolla*, *fimiseda*). (See R. F. Cain, *Univ. Toronto Stud. biol.*, 38, 1934.)

'*Polyporus abietinus* (Dicks.) Fr.'; ('Weissfäule').

= *Polystictus abietinus* (Dicks.) Fr.; (pitted sap rot of conifer wood). (See Cartwright & Findlay, 1946.)

'*Polyporus fomentarius* L.'; ('Zunderschwamm').

= *Fomes fomentarius* Karst.; (tinder bracket, tinder fungus). This fungus is rare in England but causes a heart rot of birch in Scotland (see *B.M.S. List*) and is widely distributed in Europe and America where it causes a white mottled rot of hardwoods. (See Cartwright & Findlay, 1946.)

'*Polyporus Hartigii* All.'; ('Weissfäule der Weisstanne').

= *Fomes hartigii* (Allesch.) Sacc. & Trav.; often regarded as a variety of *F. robustus* Karst. *F. robustus* is rare in Britain but occurs on the Continent where it is confined to oaks and chestnuts. *F. hartigii* attacks conifers (fir, spruce, hemlock) and trees often break off where the sporophores occur.

'*Polyporus vaporarius*'; ('Mauerschwamm').

Gäumann refers to the 'Mauerschwamm' as *P. vaporarius* without appended author (*orig.*, p. 257), as *P. vaporarius* Pers. (*orig.*, pp. 315, 403, and Index), and as *P. vaporarius* Fr. (*orig.*, pp. 438, 439). There is much confusion regarding the identity of the dry-rotting fungus referred to by Continental writers as *P. vaporarius*. It is now usually understood to be the same fungus as that termed *Poria vaporaria* Pers. The fungus which frequently causes dry rot in Britain is *Poria vaillantii* (DC.) Fr. (See Cartwright & Findlay, 1946.)

Potato aucuba mosaic; ('Aucubamosaik').

Potato aucuba mosaic virus (*R.A.M.*, 1946); *Solanum* virus 9 (Smith, *Tb.*); *Marmor aucuba* (*M.D.B.*, 1948).

Potato crinkle; (*orig.*, p. 74: '... Mischvirosen der Kartoffeln (crinkle, para-crinkle; Abb. 155) ...'; p. 274: 'Das Z-Virus ... in Mischinfektion mit dem X-Virus ein 'Crinkle' ...').

Gäumann's use of the term 'Crinkle' is a little ambiguous but seems to be in Salaman's (1930-2) sense of 'Crinkle "A"' = potato virus Z + potato virus A. Thus according to *R.A.M.* (1946) usage it would be potato para-crinkle virus + potato virus X; according to Smith (*Tb.*) it would be *Solanum* virus 7 + *Solanum* virus 1; and according to *M.D.B.* (1948) it would be *Marmor anglicae* + *Annulus dubius* (syn. *Marmor dubium* (Holmes, *Hb.*)).

Potato leaf roll; ('Blattrollkrankheit der Kartoffeln').

Potato leaf roll virus (*R.A.M.* 1946); *Solanum* virus 14 (Smith, *Tb.*); *Corium solani* (*M.D.B.*, 1948).

Potato mottle; ('Kartoffelmosaik'; 'X-Viren der Kartoffeln'; 'Kartoffel-X-Mosaik-virus').

Potato virus X (*R.A.M.*, 1946); *Solanum* virus 1 (Smith, *Tb.*); *Annulus dubius* (*M.D.B.*, 1948), syn. *Marmor dubium* (Holmes, *Hb.*). Various strains—G, H, L, N, S. (see Salaman, 1938).

Potato net necrosis; ('Netznekrose der Kartoffeln').

= Potato leaf roll virus + potato virus A; q.v.

Potato paracrinkle; (*orig.*, p. 74: '... Mischvirosen der Kartoffeln (crinkle, paracrinkle; Abb. 155) . . .'; p. 188: '... einer komplexen Virose, nämlich an Paracrinkle . . .'; p. 274: 'Das Z-Virus . . . in Mischinfektion mit dem Y-Virus das Paracrinkle . . .').

Gäumann's use of the term 'Paracrinkle' is a little ambiguous but seems to be in Salaman's (1930-2) sense of 'para-crinkle' = potato virus Z + potato virus Y. Thus according to *R.A.M.* (1946) usage Gäumann's 'Paracrinkle' would be potato paracrinkle virus + potato virus Y; according to Smith (*Tb.*) it would be *Solanum* virus 7 + *Solanum* virus 2; and according to *M.D.B.* (1948) it would be *Marmor angliae* + *Marmor upsilon* (syn. *Marmor cucumeris* var. *upsilon* (Holmes, *Hb.*)).

Potato phloem necrosis; ('Phloëmnnekrose der Kartoffeln').

See Potato leaf roll.

Potato virus A; ('A-Virus').

Potato veinal mosaic (*R.A.M.*, 1946); *Solanum* virus 3 (Smith, *Tb.*); *Marmor solani* (*M.D.B.*, 1948).

Potato virus F.

See Potato aucuba mosaic.

Potato virus X; ('Kartoffel-X-Viren').

See Potato mottle.

Potato virus Y; ('Y-Virus der Kartoffeln').

Leaf drop streak (primary symptoms), severe mosaic (secondary symptoms) (*B.M.S. List*); potato rugose mosaic (*R.A.M.*, 1946); *Solanum* virus 2 (Smith, *Tb.*); *Marmor upsilon* (*M.D.B.*, 1948) (syn. *Marmor cucumeris* var. *upsilon* (Holmes, *Hb.*)).

Potato virus Z; ('Z-Virus, das symptomlose Virus der Kartoffelsorte King Edward').

Potato paracrinkle virus (*R.A.M.*, 1946); *Solanum* virus 7 (Smith, *Tb.*); *Marmor angliae* (*M.D.B.*, 1948).

'*Pseudomonas campestris* (Pam.) E.F.S.'; ('Schwarznervigkeit, Trockenfäule, Adernschwärze des Kohles').

= *Xanthomonas campestris* (Pam.) Dowson (*B.M.S. List*) (*M.D.B.*, 1948); syn. *Phytoplasma campestris* (Pam.) Bergey *et al.*; (black rot of cabbage, cauliflower, seakale, turnip, &c. (*B.M.S. List*)).

'*Pseudomonas cerasi* Griff.'; ('... bakteriellen Gummiflusses der Süßkirschen in Nordamerika').

= *Ps. cerasus* Griffin (*B.M.S. List*); (bacterial canker and leaf spot of cherry (*B.M.S. List*)). *M.D.B.* (1948) follows Wilson (1940) in identifying *Phytoplasma cerasi* (= *Pseudomonas cerasus*) with *Pseudomonas syringae* van Hall (= *Bacterium syringae* (van Hall) E.F.Sm.; q.v.).

'*Pseudomonas citri* Hasse'; ('Citrus-Krebs').

= *Xanthomonas citri* (Hasse) Dowson (*M.D.B.*, 1948); syn. *Phytoplasma citri* (Hasse) Bergey *et al.*; (citrus canker).

'*Pseudomonas endiviae* Kotte'; ('Blattfleckenkrankheit der Endivie').

? = *Pseudomonas cichorii* (Swingle) Stapp (*M.D.B.*, 1948); syn. *Phytomonas endiviae* (Kotte) Clara; ? syn. *Bacterium formosanum* Okabe; (leaf spot of endive).

'*Pseudomonas hyacinthi* Wakk.'; ('... gelben Rotz der Hyacinthen in Holland').
= *Xanthomonas hyacinthi* (Wakk.) Dowson (*B.M.S. List*) (*M.D.B.*, 1948);
syn. *Phytomonas hyacinthi* (Wakk.) Bergey *et al.*; (yellow disease of hyacinth (*B.M.S. List*)).

'*Pseudotsuga Douglasii* Carr.'; ('Douglasie').

= *P. taxifolia* Britt.; (common Douglas fir (*S.P.N.*); Oregon Douglas fir).

'*Puccinia anomala* Rostr.'

See *P. hordei*.

'*Puccinia coronata* Cda.'; ('Kronenrost').

Syn. *P. lolii* Niels. (= *P. coronifera* Kleb.); (crown rust of cereals and grasses).

In his text Gäumann cites all three above names and also *P. lolii avenae* Erikss., but gives (*orig.*, p. 446) '*Puccinia lolii* Niels. (syn. *Puccinia coronifera* Kleb.)' and in his Index '*Puccinia coronifera* Kleb. s. *Puccinia lolii*'. He accepts Klebahn's division of Corda's species into *P. coronata* Kleb. and *P. coronifera* Kleb. (= *P. lolii* Niels.) and regards both fungi as pathogens of 'Kronenrost'. Brown (1937) was unable to confirm Klebahn's division and *P. coronifera* is usually regarded as a synonym of *P. coronata* (see *B.M.S. List*; Dickson, *Diseases of Field Crops*, 1947; &c.).

'*Puccinia coronifera* Kleb.'

See *P. coronata*.

'*Puccinia digraphidis* Soppitt.'; ('... einen Rost des Rohrglanzgrases').

Grove (1913) states that this fungus is a biological race of *P. sessilis* Schneid., having its aecidia on *Convallaria majalis* and *Paris quadrifolia*.

'*Puccinia dispersa* Erikss.'; ('Braunrost des Roggens').

Syn. *P. secalina* Grove; syn. *P. rubigo-vera secalis* (Eriks. & Henn.) Carleton (in Dickson, 1947); (brown rust of rye (*B.M.S. List*); leaf rust (U.S.A.)).

'*Puccinia hordei* Otth. (syn. *P. simplex* (Koern.) Erikss. & Henn.)'; ('Zwergrost der Gerste').

Syn. *P. anomala* Rostr.; (brown (dwarf) rust of barley (*B.M.S. List*); leaf rust of barley (U.S.A.)).

'*Puccinia lolii* Niels.'

See *P. coronata*.

'*Puccinia simplex* (Kcke.) Erikss. & Henn.'

See *P. hordei*.

'*Puccinia triticina* Erikss.'; ('Braunrost des Weizens').

Syn. *P. rubigo-vera tritici* (Erikss.) Carleton (in Dickson, 1947); (brown rust of wheat (*B.M.S. List*); leaf rust of wheat (U.S.A.)). A comprehensive account of this fungus and the disease it causes is given by K. S. Chester, *The Nature and Prevention of the Cereal Rusts as exemplified in the Leaf Rust of Wheat*, 1946.

'*Pythium palmivorum* Butl.'; ('Herz der Kokospalmen').

= *Phytophthora palmivora* Butl.; (pod rot and canker of cocoa).

'*Rhamnus Frangula* L.'

= *Frangula alnus* Mill.; (glossy buckthorn (*S.P.N.*); berry bearing alder).

'*Rhizoctonia crocorum* DC. (*Rhizoctonia violacea* Tul.).'

= *Helicobasidium purpureum* Pat.; stat. mycel. *Rhizoctonia crocorum* Fr.; (violet root rot (*B.M.S. List*)).

'*Rhizopus nigricans* Ehrenb.'; ('Fäulnis der Erdbeeren').

= *R. stolonifer* (Fr.) Lind; (mouldy fruit rot of tomato (*B.M.S. List*), strawberry, &c.).

'*Rhizopus suinus* Nielsen.'

In his *Clés des Mucorinées* (*Encyclopédie Mycologique*, No. ix, 1939) N. A. Naumov includes *R. suinus* in his group of 'Espèces qui nous sont restées inconnues'.

'*Ribes Grossularia* L.'; ('Stachelbeere').

= *R. uva-crispa* L.; (European gooseberry (*S.P.N.*)).

'*Rosa eglanteria* L.'

Included by Clapham (1946) in *R. rubiginosa* L. agg.; (sweet-brier rose (*S.P.N.*)).

'*Rosa glauca* Pourr.'

= *R. rubrifolia* Vill.; (redleaf rose (*S.P.N.*)).

'*Rosa lutea* Mill.'

= *R. foetida* Herrm.; (Austrian brier rose (*S.P.N.*)).

'*Rosa pomifera* Herrm.'

Included by Clapham (1946) in *R. villosa* L. agg.; (apple rose (*S.P.N.*)).

'*Sclerotinia ciborioides* (Hofm.) Eriks. (= *S. trifoliorum* Eriks.); ('polyphag und greift ausser *Trifolium*- auch *Medicago*-, *Onobrychis*- usw. Arten an').

= *S. trifoliorum* Erikss.; (rot of clover, lucerne, sainfoin, trefoil, &c. (*B.M.S. List*); *Sclerotinia* root rot and crown rot (*U.S.A.*)).

'*Sclerotinia cinerea* Schroet. (syn. *Monilia cinerea* Bon.); ('Polsterschimmel des halbreifen Steinobstes'; 'Blüten- und Zweigdürre und Fruchtfäule des Steinobstes'; 'Grauschimmel bei Pfirsichen').

= *S. laxa* Aderh. & Ruhl., stat. conid. *Monilia cinerea* Bon. (*B.M.S. List*); syn. *Monilinia laxa* (Aderh. & Ruhl.) Honey (Whetzel, 1945); (brown rot, blossom wilt, spur blight, and wither tip of plum, &c. (*B.M.S. List*)).

'*Sclerotinia fructigena* Schroet.'

= *S. fructigena* Aderh. & Ruhl., very rarely other than in stat. conid. *Monilia fructigena* (Fr.) Westend.; syn. *Monilinia fructigena* (Aderh. & Ruhl.) Honey (Whetzel, 1945); (brown rot and spur canker of apple; brown rot of pear, plum, peach, quince (*B.M.S. List*)).

'*Sclerotinia Linhartiana* Prill. & Delacr.'; ('... Polsterschimmel der betreffenden Früchte auf Quitten').

= *S. cydoniae* Schellenb., stat. conid. *Monilia necans* Briosi & Cav.; syn. *Monilinia cydoniae* (Schell.) Whetzel; (leaf blotch of quince (*B.M.S. List*)).

'*Sclerotinia rhododendri* Fisch.'

= *Monilinia rhododendri* (Fischer) Whetzel.

'*Sclerotinia sclerotiorum* (Lib.) Sacc. & Trott. (= *S. Libertiana* Fckl.); ('Lagerfäulen'; 'dem Erreger zahlreicher "*Botrytis*"-Erkrankungen auf Speicherorganen, Rhizomen, Zuckerrüben, Kartoffeln, usw.'; 'ein polyphager Parasit auf allen möglichen Kulturpflanzen und Gemüsen').

Although from the viewpoint of physiological pathology the diseases caused by *S. sclerotiorum* may be described as 'of the *Botrytis* type' there is, of course, no evidence that this fungus possesses a *Botrytis* conidial state. *S. sclerotiorum* (Lib.) De Bary; (*Sclerotinia* disease of artichoke, hop, tomato; *Sclerotinia* rot of carrot; stalk break of potato (*B.M.S. List*)).

'*Solanum Lycopersicum* L.'; ('Tomate').

= *Lycopersicon esculentum* Mill.; (common tomato (*S.P.N.*)).

'*Sorosporium Reilianum* (Kühn) McAlp.'; ('Hirsebrand').

= *Sphacelotheca reiliana* (Kühn) Clint.; (head smut of maize and sorghums).

'*Sphaeropsis malorum* Peck'; ('Zweigkrebs und Schwarzfäule der Äpfel').

Imperfect state of *Physalospora obtusa* (Schw.) Cooke; (leaf spot and black rot of apple (*B.M.S. List*)).

'*Suaeda Moquini* Greene.'

= Var. of *S. fruticosa* Forsk.; (alkali seepweed (*S.P.N.*)).

'*Thekopsora areolata* (Fr.) Magn.'; ('Rost der Traubenkirsche').

= *Thekopsora padi* (Magn.) Dietel; syn. *Pucciniastrum padi* Dietel; (rust of bird cherry).

'*Thielavia basicola* (B. & Br.) Zopf'; ('Wurzelfäule, Fusskrankheit oder Wurzelbräune des Tabaks').

There has been much confusion between the two fungi, *Thielavia basicola* Zopf and *Thielaviopsis basicola* (Berk. & Br.) Ferraris. The former is a saprophytic member of the Eurotiales which forms its perithecia when grown in mixed culture with *Thielaviopsis basicola*. The latter is a parasitic member of the Moniliales which causes black rot of pea, and root rot of tobacco. (See G. B. Lucas, *Phytopathology*, **38**, p. 16, 1948.)

'*Tilletia laevis* Kühn'; ('Weizensteinbrand').

= *T. foetida* (Wallr.) Liro; (bunt or stinking smut of wheat). A common agent of bunt in North America but not recorded on growing crops in Britain, where this disease is caused by the closely related species *T. caries* (DC.) Tul.; (syn. *T. tritici*, q.v.).

'*Tilletia tritici* (Bjerk.) Wint.'; ('Weizensteinbrand').

= *T. caries* (DC.) Tul.; (bunt or stinking smut of wheat).

Tobacco mosaic; ('Tabakmosaik'; 'Tabakmosaikvirus').

Tobacco mosaic virus (*R.A.M.*, 1946); *Nicotiana virus* 1 (Smith, *Tb.*); *Marmor tabaci* (*M.D.B.*, 1948). Numerous strains and suspected strains; for synonymy see *R.A.M.*, 1946.

Tobacco ring spot; ('Ringfleckenvirus'; 'Ringfleckenkrankheit'; 'Ringfleckigkeit des Tabaks').

Tobacco ring spot virus (*R.A.M.*, 1946); *Nicotiana virus* 12 (Smith, *Tb.*); *Annulus tabaci* (*M.D.B.*, 1948).

Tobacco ring spot mosaic; ('Ringfleckenmosaik').

Gäumann apparently uses 'Ringfleckenmosaik' (*orig.*, p. 293) as a synonym of tobacco ring spot ('Ringfleckigkeit') and not in the sense of Valteau & E. M. Johnson's (1930) tobacco ring mosaic virus which is a strain or suspected strain of tobacco mosaic virus (see *R.A.M.*, 1946).

Tobacco ring spot necrosis; ('Ringfleckennekrose des Tabaks').

Gäumann apparently uses 'Ringfleckennekrose' (*orig.*, pp. 60, 371, Abb. 223) as a synonym of tobacco ring spot ('Ringfleckigkeit') and not in the sense of Thung's (1935) tobacco ringspot necrosis which is a strain or suspected strain of tobacco mosaic virus (see *R.A.M.*, 1946).

Tomato mosaic; ('Tomatenmosaik').

See Tobacco mosaic.

Tomato streak; ('Streak-Virus auf Tomatenblättern'; 'Streak-krank Tomaten').

See Tobacco mosaic.

'*Trametes pini* (Brot.) Fr.'; ('Ringschale der Fichten und Kiefern').

T. pini (Fr.) Fr. is accepted by most English workers but in America the

fungus is usually called *Fomes pini* (Thore) Lloyd; (heart rot of Scots pine (*B.M.S. List*); red ring rot, ring scale, red heart or pecky rot of most conifers (*U.S.A.*)).

'*Trametes radiciperda* Hartig (syn. *Polyporus annosus* Fr.)'; ('... trockenen Rotfäule oder Stockfäule der Fichten und des stehenden Kiefernholzes').

= *Fomes annosus* (Fr.) Cooke; (annosus butt rot (*B.M.S. List*); Fomes root rot, spongy sap rot, brown root rot, butt rot (*U.S.A.*)).

'*Tranzschelia pruni spinosae* (Pers.) Diet.'; ('Zwetschgenrost').

= *Puccinia pruni-spinosae* Pers.; (apricot and plum rust, cluster cup rust of anemone (*B.M.S. List*)).

'*Trichoderma lignorum* (Tode) Harz'; ('Fäulniserreger auf Wurzeln, Früchten, usw.').

= *T. viride* Pers. ex Fries; syn. *Eidamia viridescens* Horne & Williamson; probably an imperfect state of *Hypocrea* spp.; (a common soil fungus which causes moulding of damp wood, &c.).

'*Tylenchus tritici* (Steinb.) Bast.'; ('Weizenälchen').

= *Anguillulina tritici* (Steinbuch) Gervais & v. Beneden.

'*Ulmus scabra* Mill. (= *U. montana* With.).'

= *U. glabra* Huds.; (Scotch or wych elm (*S.P.N.*)).

'*Uromyces fallens* (Dam.) Kern'; ('Kleerost').

U. trifolii (Hedg. f.) Lév.; (clover rust (*B.M.S. List*)).

'*Ustilago levis* (K. & S.) Magn.'; ('Hartbrand des Hafers').

= *U. kolleri* Wille; (covered smut of oats (*B.M.S. List*)).

'*Ustilago zeae* (Beckm.) Ung.'; ('Maisbrand'; 'Beulenbrand des Mais').

Syn. *U. maydis* (DC.) Cda.; (corn (maize) smut).

'*Verbena erinoides* Lam.'

= *V. laciniata* Briq.; (moss Verbena (*S.P.N.*)).

'*Vibrio cholerae asiaticae*.'

= *Vibrio comma* (Schröter) Winslow *et al.* (*M.D.B.*, 1948).

'*Vincetoxicum officinale* Mönch'; ('Schwalbenwurz').

= *Cynanchum vincetoxicum* Pers.; (white swallow-wort (*S.P.N.*)).

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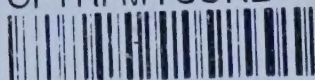
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